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THE INNERVATION OF STRIPED MUSCLE-FIBRES AND LANGLEY'S RECEPTIVE SUBSTANCE.[1]

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Let me try to give you in this lecture in broad outlines the results of recent investigations on the mode of innervation of the striped muscle-fibres of vertebrates, the origin of the nerve-fibres which reach the muscle-fibres, their mode of ending on them, the manner in which the nerve-endings are connected with the muscle-fibres and the path by which the stimuli arriving by the nerves may reach the contractile substance of the muscle-fibre.

In the time at my disposal I must necessarily treat the subject somewhat dogmatically, and I fear that I shall not be able to do much more than express my own views about this subject without giving you a full historical account of the questions or stating the authorship of the large number of investigations carried out in this field and bearing on the innervation of the striped muscle-fibres of vertebrates and in general on the relation of the nerve-endings to their surroundings, on which these views are founded.

For a morphologist like myself it is only natural that morphological details will come in the first place to call our attention. But it seems to me that in general (apart from questions of phylogeny and comparative anatomy) morphological details derive their full value in the first place from the physiological and functional insight they are able to furnish us. For only when we are able to give a physiological interpretation of a given morphological detail can we say that we understand it, that it has given us some new insight into the ways of Nature.

But first let me state some general principles.

It cannot be denied that animal histology nowadays is in a period

[1] Lecture delivered at the University of London, on February 16, 1921.

of unrest, of evolution, of reform, that it is passing through a crisis, which is shaking its very foundations. The old conception of the cell as an elementary organism, established as the basis of all our researches after years of strife and an incredible amount of labour—this conception of the cell, on which generations of our teachers had built up the entire splendid structure of our knowledge of the animal body and its component parts, must be revised. And this process of revision has met with frequent and often serious opposition.

Obviously the cytologist, when studying and analysing the structure of the smallest elements that build up the living body, the cells and their derivations, is strongly tempted to regard these cells as units, as indivisible self-propagating independent elementary organisms, originated in a direct line from the dividing egg-cell and remaining independent units until their death or the death of the organism which they helped to build up.

This is not to be wondered at. It is not only that we have learned, since the time of Robert Brown, of Schleiden and Schwann, of Max Schultze, Huxley, Ranvier and Virchow, that all living bodies are composed of those small units, the cells, and their differentiation products. But afterwards there has been brought to light inside these cells themselves a distinct organization, a world in itself, a number of independent self-propagating structures, the nucleus, the amyloplasts, the tonoplast of de Vries and Went, the chlorophyll-bodies, the elaioplasts of Wakker, the centrosome, the mitochondriæ and their differentiation products, and in the nucleus the chromosomes with their outfit of chromomeres, which during the last years have become of such enormous significance, especially through the work of Morgan and his school on the physical basis of hereditary phenomena.

And we are here still standing on the threshold. Every new line of research started reveals to us unknown wonders of organization of the cells. But, on the other hand, when by the use of the more and more accurate and subtle methods of present-day histology, we penetrate farther and farther into that wonderful organization of the cells, the cell itself loses a part of its independence as an elementary organism. Many of them we see in the course of development losing their boundaries, and fusing together to form a syncytium. And even when, as seems to be the case in the myocardium of mammals, this syncytium in a later period of development is again differentiated into cells, even here the lines of junction of the cells are bridged across by the muscle-fibrils, and we are by no means sure

that the cellular elements, which we are able with tolerable accuracy to make out in the full-grown myocardium, are the selfsame elements which were seen to constitute the heart muscle tissue before the coalescing period.

The cross-striated voluntary muscle-fibres with their immense number of nuclei, formerly supposed generally to be formed by the elongation of a single enlarged cell, have now been shown to be the product of more than one cellular element.

In the first place they are built up by the joining together, end to end, of a number of cells of the muscle-plate, so as to form a syncytium, within which the striated fibrils make their appearance. But, secondly, even more than one segmental muscle-plate may contribute to the formation of a single muscle-fibre, as Godlewski and Sunier have shown and Agduhr was able to prove by their mode of innervation, and thirdly, as Sunier has shown, even mesenchymatous elements may take a part in the formation of the muscle-fibres.

As to the smooth muscle-cells, what we know about their development tends in the same direction. According to the observations of McGill the smooth muscle-cells of the alimentary canal of the pig, seemingly so distinctly separate as true cellular organisms in the adult animal, are developed from a syncytium of mesenchymatous cells surrounding the entoderm tube. Here too the myofibrils developing in their protoplasm need not be confined to the limits of a single-cell territorium, but may extend over two or even a number of cells. In the adult smooth muscle-cells the intercellular substance is bridged across by filaments passing from cell to cell, so that even the fully formed smooth muscle-tissue retains something of its syncytial character.

The same holds true for the follicle-cells of the ovary, for many forms of connective tissue, and, last and not least, for the elements of the nervous system. For, according to the observations of Held, during their development they seem to pass through a syncytial period that leaves them by no means the selfsame elements that went into it. And during their later development and in the course of the process of regeneration, as we shall see more clearly later on, the growing axons make use of conducting cells and other "mesenchymatous" or "nervous" elements to reach their destination.

In short, wherever we study the elements of the tissues more closely, and especially histogenetically, we get the impression that the cell-lineage of the cellular elements of the full-grown body is in

most cases by no means clear. Everywhere new elements are being formed, not always entirely independent of each other, and out of a syncytial stage these elements appear as new cells, independent of the old ones, which originally went into it.

Viewed in this light, the relation of the cell to the organism as a whole, to the individual, is changing too. The embryological experiments of recent years, for instance, the experiments in which two egg-cells were made to fuse together to form one giant individual of normal form and proportions, the brilliant experiments of Fischel, of Spemann and Lewis on the formation of the eye-lens out of different tissues, the experiments on regeneration and so many more splendid achievements of the most subtle latter-day operative technique, have placed this relation entirely on a different footing. We are everywhere struck with the exquisite harmony of the living organisms, a harmony between the different tissue elements, which demonstrates the domination of the individual over the elements which compose it, the cells.

And it is this harmony which alone can guarantee that equilibrium of the different parts of the organism which Nature is always and everywhere trying to establish or to re-establish when it was lost.

The conception of a struggle for life of the cells in the organism, worked out by Roux years ago to form the basis of his theory of functional adaptation as an analogon to the idea of a struggle for life in Nature, which Darwin revealed to us by turning upon it the full light of his genius, cannot be true, because it does not sufficiently take into account this harmony of the organism.

In Nature, the individual itself is fighting for its life and for the maintenance of its species. In this fight every individual is destroying remorselessly everything that is weaker, and only the fittest survive in the struggle. What we call the harmony of Nature is founded on our own artistic view of the things around us. In Nature itself it is only the elementary forces and energies that exist, which together build up that mighty symphony that the lover of Nature hears everywhere around him. When we speak of the unity in Nature, either we unconsciously transfer the idea of the individual organism to the conception of the universe, or we feel behind the forces and phenomena of Nature a Divine power, before which we can only bow our heads in profound submission, without trying to explain it.

But in the organism this harmony is a distinct reality. Every organism is a unity in itself, and the elements which compose it are

in the first place a part of the whole, deriving their full value not from themselves, but from the individual to which they belong. The individual, the organism as a whole, dominates the cells which compose it. It blends them together, so to speak, subjugates them, to form that unity of the living organisms, that harmony of their organization, which is the greatest wonder and mystery of Nature, and fills us with such a profound admiration.

It is not to be wondered at, that among the different parts of the organism it is the nervous system, which, if this conception is true, must in the first place exhibit phenomena in harmony with this view. And we do not look in vain.

It is not only that the neurone theory, the nerve-cell a distinct unity, even embryologically, histologically, functionally, a separate, independent organism, a true cell of the old school, has to be revised. It was shown by Held and his school, and afterwards I was able to confirm his statements in a series of admirable preparations by Dr. Heringa, that the fibres of the neuroblasts do not run free in the interstices between the cells, but that they everywhere follow distinct protoplasmatic paths either of migrant medullary elements or of mesenchymatous cells, until they reach their destination. In the adult animal too we never find free "naked" nerve-fibres running in the interstices between the cells of the tissues, not even inside the epithelium, as I hope to show you later on. Wherever we find a suitable object (as for instance the corpuscles of Grandry or of Meissner, the organ of Eimer in the mole, the muscle-spindles of the cross-striated muscles of different mammals, &c.) that enables us to study the course of the end-ramifications of the nerve-fibres, we can state definitely that nowhere does a neurofibrillar strand run free, independent of the surrounding elements. And in regeneration the same fact stands out clearly, amidst the often bewildering pictures we get in our preparations of the exuberance of the regenerating nerve-fibres.

Nor is this all. As I hope to show you in the course of this lecture, during the process of nerve regeneration after a nerve has been cut there exists, or is established, a perfect harmony between the elements of the different tissues which build up the path that has to be followed by the regenerating nerve-fibres. It is not only the nerve-fibre that is growing out and seeking its way to re-attain its original destination, but all the surrounding elements, the sheath-cells, the connective-tissue cells, the elements of the original end-

organs (either the sensory end-organs or the muscle-fibres) are seen taking a part in the regeneration process in perfectly harmonious co-operation, none remaining passive, none predominating in its action. And so the re-establishment of the nervous function in regeneration is not due to the simple outgrowth of the axon processes of the nerve-cells as independent units, but to the combining together of the forces of all the surrounding elements, to restore the harmonious equilibrium of the organism, which was disturbed by the degeneration of the peripheral end of the severed nerve.

But now to return to the subject of our lecture, the innervation of the striped muscle-fibres.

About the general structure of the motor end-organ of the higher vertebrates I need not enter into detail here. We know that the end branches of the myelinated motor nerve-fibres having reached

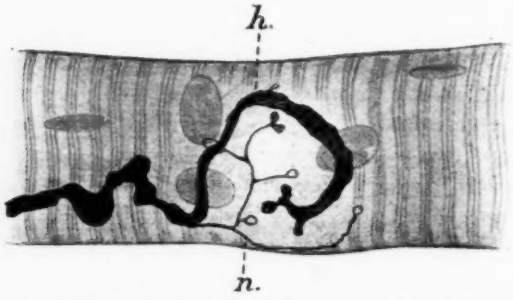

FIG. 1.—Normal motor endplate on a striped muscle-fibre of the hedgehog.

the muscle-fibres pass through the sarcolemma. The neurilemma of the nerve-fibre fuses with the sarcolemma and the axis-cylinder of the fibre is seen ending in a close terminal ramification with varicose expansions on its branches, which when appropriately fixed and stained show a fibrillar structure with reticulated or loop-like expansions. Having passed the sarcolemma, this ramification of the neuro-fibrillæ lies embedded in a layer of granular protoplasm (the sole), which is collected in a small mass at the place of the nerve-ending, and contains a number of nuclei.

Is this hypolemmal position of the nervous end-ramification only to be regarded, as Heidenhain contends, as an arrangement to attach the end-organ more strongly to the muscle-fibre ("Verankerung") and to prevent it from being torn from the muscle-fibre by a violent

contraction? I do not think so. There is no stronger mode of attachment of one element to another imaginable than that of the ends of the muscle-fibre to the tendon. And yet it is easy to demonstrate that, contrary to the well-known views of Oscar Schultze, the sarcolemma extends over the end of the fibre, the tendon-fibrils are attached to the outer surface of the sarcolemma (van Herwerden, Péterfi), and there is no actual continuity between contractile substance and tendon. Why then should such a continuity between the nerve-ending and the muscle-fibre be needed, only to prevent the nervous end-plate from being torn from the muscle-fibre by its contraction? In my opinion this hypolemmal position is only another example of the insufficiency of the old cellular conception, of the continuity of the different cellular structures with each other. And, as we will see further on, it is here of the utmost importance for the regular function of the motor end-organs.

FIG. 2.—Intraprotoplasmic ending o a nerve-fibre on a smooth muscle-fibre of the musculus ciliaris of the human eye.

In this connection it may be of interest to state, that in smooth muscle-cells the same peculiarity is met with. Here too the terminal nerve-fibrils pass into the cell, ending within the protoplasm with loop-like expansions, which often are seen lying so close to the nucleus of the muscle-cell, that they even make an indentation and thus are found lying in a shallow cavity in the side or on the top of the elongated nucleus, thereby proving their intraprotoplasmic position (fig. 2).

About the form of the neurofibrillar end-ramification I will say here a few words only. We know that the muscle-fibres of the different

classes of vertebrates possess characteristic forms of nerve-endings, from the curious spade-like expansion on the muscle-plates of amphioxus to the complicated ramifications of the mammalian motor nerve-endings. But this is not all. In every animal the end-ramification seems to represent a distinct type, so that the motor nerve-endings of a rabbit may be distinguished from those of a hedgehog or a bat; and, for instance, the nerve-endings on the muscle-fibres of a crow are of a coarser structure and form than those of the canary. Even in so

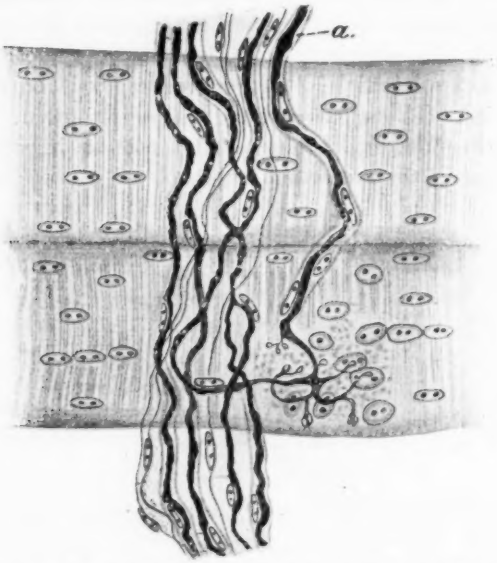

FIG. 3.—Regeneration of the motor endplates of the hedgehog, sixty-two days after the section of the nerves.

insignificant a detail as the form of the neurofibrillar network the nerve-ending appears to be in exquisite harmony with its surroundings, a part of the whole and not a thing in itself. A striking example of this harmony is again furnished by the regeneration process. When a motor nerve has been severed, after a certain lapse of time functional continuity between the cut ends may become re-established by an outgrowth of new fibres from the central stump, the newly sprouting fibres finding their way through the scar tissue between the cut ends of the nerve. After having traversed this tissue, they pass towards the periphery along the course of the degenerated fibres, the sheaths of

which serve as guides for the down-growing axons, until they reach their destination, the muscle-fibres deprived of their innervation by the cutting of the nerve. Then there are formed on those muscle-fibres end-ramifications (fig. 3); these show an exuberance of delicate nerve-fibrillæ, growing out between and on the muscle-fibres, forming end-organs of the most bizarre and complicated structure, entirely different from the normal motor-endings, of which they are the substitutes, especially in their exuberance of form and abundance of ramifications. In this way functional continuity is re-established, nerve-impulses are able to reach the muscle-fibres, contractility and voluntary movement is restored.

But the process of regeneration is not finished yet. When we keep the animal alive some months after functional activity is restored, we find the regenerated motor nerve-endings again changing their form. The complicated, aberrant end-plates, with their exuberance of branches, so totally unlike the normal end-plates, are gradually disappearing. Superfluous expansions of the neurofibrillar structures seem to be drawn in. New smaller branches are formed, and by degrees we see, in the course of a few months, everywhere reappear the normal form of the nerve-endings, characteristic for the species studied, until the equilibrium of form between the different elements constituting the muscle is restored. Only then does the regeneration process come to an end. It is this equilibrium of form, this perfect harmony between the elements, that Nature aims at, and not only the restoration of function.[1]

To this end, as I have mentioned before, all the elements of the surrounding tissues are acting and striving together. It is not only the nerve-fibre that is growing out and trying to reach its original destination. The scar tissue developing between the cut ends of the nerve, especially in the beginning of the regenerative phase, is built up of delicate, elongated, branched cells, fused to form a syncytium; about these cells we are not sure whether they are entirely of nervous origin (cells of the nerve-sheaths, lemmoblasts), or derived from the connective tissue, of mesenchymatous origin. These syncytial cells are found to enclose the growing axons, direct them towards the peripheral end of the nerve, and so form a conducting tissue for the outgrowing fibres. The delicate neurofibrillar strands are, as far as can be made out, always

[1] For further details in these matters I may refer the reader to the article: "Nervenregeneration und anverwandte Innervationsprobleme," in the *Ergebnisse der Physiologie*, xix Jahrgang, 1921, where the literature bearing on the subject is to be found.

found inside these elements, running intraprotoplasmically from one cell of the syncytium to the other, until they reach the peripheral nerve-end. Here again a syncytium is formed, the cells of the neurilemmal sheath of the degenerated nerve-fibres having proliferated and fused together into long protoplasmic strands occupying the old nerve-sheaths. The regenerating outgrowing nerve-fibres are drawn towards these protoplasmic strands or conducted to them by the elements of the scar tissue ("hodogenesis" of Dustin), and enter them, here again running intraplasmically. When the regenerating nerve-fibres have traversed the peripheral nerve-end, and have reached their destination, they again are drawn towards the muscle-fibres. We must suppose that the sarcoplasmic soles of the old degenerated endings exercise a neurotropic and neurocladic influence upon the ingrowing nerve-fibres,

FIG. 4.—Formation of collateral outgrowths of the sheath-cells of a regenerating motor nerve-fibre on a muscle-fibre of the hedgehog.

owing to which they are drawn towards them, enter them and immediately form a number of branches. These branches, collateral and terminal, form together the new nerve-endings described above. It is obvious that the most valuable phase of the regeneration process is the formation of these new end-organs, for without them no functional continuity ever can be obtained. And it is wonderful to see how all the forces of the organism are called into play here, acting together in harmonious co-operation to restore the lost equilibrium. It is not only that the outgrowing nerve-fibres are conducted towards their destination by the protoplasmic strands of the old nerve-sheaths, and that the sarcoplasmic soles attract them and cause them to form their terminal ramifications on the muscle-fibres. First, we see the cells of the nerve-sheath of the nervous plexus between the muscle-fibres beginning to proliferate, to form collateral protuber-

ances, outgrowths, reaching the muscle-fibres (fig. 4), establishing new connections into which and through which the outgrowing nerve-fibres pass to their destination. Secondly, the connective-tissue elements appear to build up a new syncytial conducting tissue throughout the atrophied muscle to the same purpose, the formation of new ways for the outgrowing nerve-fibres. It is true that this last-named proliferation of the connective-tissue elements is by no means easy to follow with sufficient clearness in the sections through the close-packed muscle-fibres of the common muscles. But let us examine places where we find the muscle-fibres more loosely arranged, as is the case, for instance, in the muscle-spindles. Here, as it was described so well by Sherrington, we find the axial spindle-fibres completely surrounded by a large lymph space, bridged across and partially subdivided in

FIG. 5.—Conducting tissue of the periaxial space of a muscle-spindle of the hedgehog, with neurofibrillar strands.

many points by extremely tenuous membranes, filaments, and strands of syncytial connective-tissue elements. And here we can state definitely, not only that the end-ramifications of the normal nerve-fibres are everywhere enclosed within the protoplasm of these mesenchymatous cells (fig. 5), but that during the degeneration phase of the regeneration process these syncytial cells proliferate, branch and form numerous new connections, so that a typical conducting syncytium is formed which catches up the ingrowing fibres, encloses them and enables them to reach their destination, the axial muscle-fibres.

And now, after having studied these things in the muscle-spindles, when turning again towards the ordinary muscle-fibres, we detect everywhere traces of the same process going on here, of the same conductive tissue forming between the muscle-fibres. And I can imagine no other

process which affords us such a striking example of the harmonious co-operation of the elements of the different tissues as subordinate parts of the whole, to reach a given end, the restoration of the equilibrium of the organism, than this mode of regeneration of the motor nerve-endings.

The most prominent feature of the motor nerve-endings of vertebrates (and, as far as they have been studied, the same holds true for those of invertebrates) is the expansion of the neurofibrillar structure. As soon as the axis-cylinder passes through the sarcolemma we see the bundle of neurofibrillæ, after a momentary constriction, broaden out, disperse and expand into branched networks or looplike structures. A typical example of this expansion is exhibited in the figures. Now it is well known that ever since these neurofibrillæ were described by Apathy, who succeeded in staining them in his preparations with the most wonderful accuracy, not only their arrangement, connections and other morphological details have been under constant discussion, but morphologists and physiologists alike have been discussing the question of the function which we have to ascribe to these peculiar fibrils, which are found without exception in all parts of the ganglion-cells and their processes. Apathy himself regards them as the sole conductors of the nerve-impulses, and, in the lecture delivered at Cambridge in 1898, gave a number of arguments in support of this conclusion. In later years it was chiefly Bethe who took up the idea and carried out several interesting experiments to furnish proofs for it. A number of investigators agreed with him. Others, such as Wolff, von Lenhossek, Verworn, Jenkins and Carlson, Cajal, are convinced that the nervous impulses are also conducted by the neuroplasmic substance enveloping the neurofibrillar structure, either by the two substances together or by the neuroplasm alone. In this case the neurofibrillæ only play the part of supporting filaments of the nervous elements. It would lead us too far to review all the different statements and arguments of the numerous authors who have contributed to the question under discussion. In my opinion there is only one conclusion to be drawn from the different observations on this point, and that is, that we have to see in the neurofibrillæ the actual conductors of the nerve-impulses.

But if this assumption be true, we are confronted with a real difficulty. In general there seems to be no connection between the neurofibrillar structure of the motor-endings and the contractile substance of the muscle-fibre. Even Kühne, who advocated so strongly the hypolemmal position of the motor-endings, pointed out (in the Croonian

Lecture) that properly stained hypolemmal branches terminate usually in rounded ends sharply marked off from the muscle substance. According to Langley, even in the simplest case, that of the nerve-endings of amphibia, where the ramifications spread over a considerable length of the muscle-fibre, the substance which has been described as continuous with the endings is clearly the sarcoplasm of the muscle, which stretches throughout the muscle-fibre, and not the contractile substance itself. And indeed, as far as my experience goes, I know of only one case in which the neurofibrillar structure (or something akin to it) is found to be continuous with the myofibrillæ. That is in amphioxus, where I found the extremely delicate terminal meshes of the neurofibrillar network to be continuous with the anisotropic discs of the muscle-plates, each anisotropic disc receiving a small branch

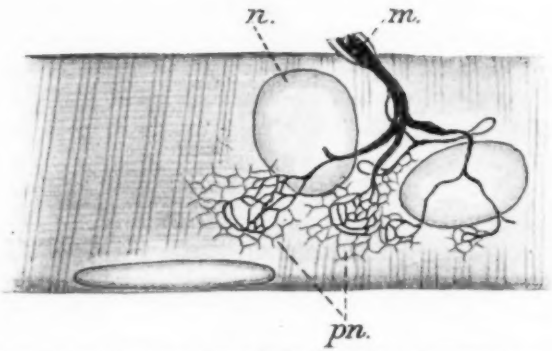

FIG. 6.—Normal motor endplate of the bat, with periterminal network; *pn.* = periterminal network; *n.* = nucleus of the sole.

from the neurofibrillar structure running along it. In all the other vertebrates there lies, when studied in ordinary preparations, a stretch of undifferentiated granular sarcoplasm between the terminal ramifications of the neurofibrillæ and the contractile substance, the myofibrillæ.

But let us study these things in carefully stained and differentiated Bielschowsky preparations, in which not only the neurofibrillar network stands out very clearly, but the adjoining tissues and elements have taken the stain, too, so as to be visible in the sections. Then there appears in the sarcoplasm surrounding the neurofibrillar end-ramifications a reticular differentiation, extremely delicate, on one side attached to the neurofibrillar ramifications, on the other side losing itself in the sarcoplasm. In my first publication I proposed for it the name of

"periterminal network," and by this name I will call it in the following pages. Enclosed in the finely granular sarcoplasm we find this periterminal network as extremely delicate fibrils, forming a reticulum with small meshes and somewhat thickened knots, which is continuous with the neurofibrillar ramifications (figs. 6, 7). Especially around the end-loops and end-nets of this structure the meshes of the periterminal network are very distinct; they appear here either as small regular, polygonal meshes of about the same size, or those surrounding somewhat larger end-loops or end-reticula of the neurofibrillar structure are of somewhat larger dimensions, slightly elongated, crescent-shaped or cup-like in appearance. But even where the innermost meshes, surrounding

FIG. 7.—Normal motor endplate on a muscle-fibre of the m. obliquus sup. of the cat, with periterminal network, seen "in profile."

the end-loops of the neurofibrillar structure, appear in this way, the second layer of meshes generally shows the polygonal, regular form, mentioned above. There where the reticulate neurofibrillar end-loops are not spread out in one plane, but are three-dimensional, more or less pear-shaped, the meshes of the periterminal network are seen surrounding them on all sides.

But it is not only with the end-loops and end-reticula of the neurofibrillar structure that the periterminal network is found to be in connection; it may be seen as clearly surrounding the more or less varicose branches of the motor-ending, and here too it is always found in close connection with the neurofibrillar structure (fig. 7). Often, when examined under the highest power, the neurofibrillar strands give the impression

of being covered with extremely tenuous spines. With these tiny spines the fibrils of the periterminal network are seen to be in connection. The meshes of the periterminal network appear to be continuous throughout the sarcoplasm of the sole, and although it is wont to take a stronger stain in the immediate neighbourhood of the axial neurofibrillar ramification, in several cases I could follow the fibrils of the periterminal network throughout the sarcoplasmic sole to the myofibrillæ; here they seem to pass into the anisotropic discs of the myofibrillæ, and to form an extremely delicate network throughout the whole thickness of the discs (fig. 7).

As to the morphological value of the periterminal network I need not enter into details here. The time at my disposal prevents me from going too far into historical details. Suffice it to say that traces of this network seem to have been seen by Retzius, Koelliker and Rollett, that it makes the impression of being an element *sui generis* and that in my opinion it is not identical with the reticular differentiations inside the muscle-fibre described by Veratti, Cajal-Fusari and Holmgren.

The periterminal network is an element *sui generis*. But is it to be regarded as of a neural nature, a continuation of the neurofibrillar structure, or is it a differentiation of the sarcoplasm itself? This is a difficult question to answer. It is always found in close connection with the neurofibrillar structure. During the development of the motor end-plates it is always found to appear as a continuation of the neurofibrillar structure. Even in properly stained sections it always takes the darkest stain where it is attached to this structure, and it is often only visible in this part of the sarcoplasm and gradually disappears from view farther on. And yet its features are so different from the neurofibrillar structure, and it gives so clearly the impression of a protoplasmic reticulum, that I do not hesitate to assert that in my opinion it is of protoplasmic (sarcoplasmic) origin, but that it develops only in connection with and under the influence of the neurofibrillar structure of the motor nerve-ending. This is important for the accurate conception of its physiological and functional significance.

An interesting point is its behaviour during degeneration and regeneration of the motor nerves. The most characteristic feature of the degeneration of the motor nerve-endings, after cutting of the nerve, is the gradual disappearance of the neurofibrillar network. Within a few hours after the operation the fibrils begin to swell, to fuse together, and the mass thus formed, in the course of one or two

days breaks up into portions and disappears. During the first stages of the process of degeneration the periterminal network may still be distinguished, but it disappears from view together with the neurofibrils, and only the granular sarcoplasm of the sole remains visible. When, after a lapse of time, the regenerated motor nerves again reach the muscle-fibres, their ends ("cônes de croissance") pierce the sarcolemma, come again in contact with the sarcoplasm and develop into new nerve-endings. As soon as the muscle-fibre is reached and the first neurofibrillar loops have entered it, the periterminal network makes its reappearance (as is shown in fig. 8) as a reticular

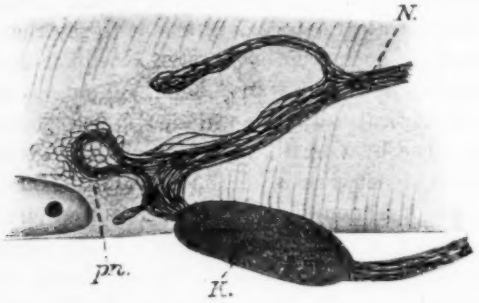

FIG. 8.—Regeneration of a motor endplate of the hedgehog, with formation of the periterminal network (*pn.*); *K.* = nucleus.

differentiation of the sarcoplasm surrounding the neurofibrils, at first only in the immediate neighbourhood of the neurofibrillar strands, but gradually spreading and growing out. So its disappearance and its reappearance are absolutely connected with and dependent on the presence or absence of the neurofibrillar structure.

All this seems to indicate that the periterminal network (whether as a sarcoplasmic reticulum or a continuation of the nervous structure) is intimately connected with the functions of the neurofibrillar structure of the motor-endings. And thus, when we have every reason, as I have mentioned already, to regard the neurofibrillæ as the actual conductors of the nerve-impulses, the assumption seems to be justified that the neurofibrillar structure, not being continuous with the contracting substance of the muscle-fibre, the impulse is carried on by the periterminal network and in this way transmitted to the contracting substance of the muscle-fibre, the myofibrillæ.

This may be brought into connection with some well-known physiological facts. In his brilliant Croonian Lecture for 1906, Langley has shown that there is every reason to suppose that in the case of a connection of the peripheral end of an efferent nerve with a cell there must be present in the cell one or more substances (which Langley calls "receptive substances") capable of receiving and transmitting stimuli and capable of isolated paralysis. According to Langley it is this substance which is stimulated or paralysed by poisons ordinarily taken as stimulating or paralysing nerve-endings (curari, nicotine, strychnine, and others). Probably not only the function of reacting to numerous chemical bodies, but also the special liability of both afferent and efferent nerves to fatigue, must be transferred from the nerve-endings to the same constituent of the cell. The nerve-ending itself is, according to Langley, physiologically not essentially different from the nerve-fibre. Therefore the special action of the poisons mentioned above must be due to the presence of one or more receptive substances in the cell. These substances are, Langley considers, radicles of the protoplasmic molecule, which reminds us of the haptophoric groups of the "Seitenketten" theory of Ehrlich; and indeed, I think that the general opinion amongst physiologists is, that a morphological substratum of the receptive substances (or the "neuroplasmatische Zwischensubstanz" of Asher) is not to be looked for in histological preparations. And not only physiologists. Even in morphological work, as for example the study of Sutton on the development of the neuro-muscular spindle in the extrinsic eye-muscles of the pig (1915), the conclusion is drawn, that until now there does not exist a morphological basis for the receptive substance.

And yet the later work of Langley and the work of Keith Lucas on the different behaviour of the neural region of the muscle-fibre, and the aneural region as regards the action of poisons, lend support to the view that such a substance exists in proximity to the nerve-endings in muscle. And it was shown by Noel Paton and Findlay in 1916 that after the efferent nerves have been cut and the nerve-endings have degenerated, the receptive substance in the muscle-fibre seems to degenerate secondarily to the neural endings. It seems to remain unaltered for some time after the nerve has ceased to act, and in some cases have its excitability modified during the process of degeneration of the nerve; after some time, however, it degenerates also.

Now all this seems to me to point to the conclusion that we may look for a well-defined, sharply localized morphological basis for the

receptive substance, that it has to be looked for inside the sarcoplasm, between the nerve-ending and the contractile substance, and that all the conditions for it are satisfactorily fulfilled by the "periterminal network" described above. It lies embedded in the sarcoplasm and indeed seems to be a part of it, it is in close connection with the neurofibrillar structure of the nerve-ending and is especially visible in the immediate neighbourhood of it, but extends to the contractile substance. It has a morphological aspect different from that of the neuro-fibrillar structure of the nerve-ending, and seems to be of a finer texture in highly developed active muscle-fibres (as the extrinsic eye-muscles of the cat for instance) than in the muscle-fibres of slower action (as the muscles of the tongue of the hedgehog). And last not least, it degenerates secondarily to the degeneration of the motor-endings, and again makes its appearance as soon as the regenerating nerve-fibres have reached the muscle-fibres and are forming new end-ramifications in the sarcoplasma.

But, as a physiological conception, the receptive substances of Langley are not confined to the striated muscle-fibres. And indeed, one is tempted to ask whether in every case of a synapse, at least of a connection of a nervous end-ramification with a peripheral element, either of an efferent or an afferent nature, a receptive substance, capable of receiving or transmitting stimuli, has not to be localized as an intervening medium between the undifferentiated protoplasm and the neurofibrillar structure of the nerve-ending.

There are several histological phenomena that seem to point in this direction. In the first place I have to mention the connections of the sympathetic nerves with the striated muscle-fibres. Some years ago it was shown that besides the endings of the spinal motor nerves on the muscle-fibres each fibre receives a branch of bundles of fine non-medullated nerve-fibres running between the muscle-fibres. These fibres clearly belong to the automatic nerve-system. For it could be shown that when the eye-muscle nerves (for instance the trochlearis nerve) are sectioned as near as possible to their origin from the mid-brain, before they receive a branch from the autonomic nerve-plexus, the motor nerves and their endings and the sensory nerves degenerate and disappear, but the non-medullated sympathetic nerve-fibres and their endings on the muscle-fibres remain unaltered. Later on it was shown by Dusser de Barennes and Boeke for the trunk muscles, and by Agduhr for the muscles of the extremities, that, when the spinal roots are cut and the spinal ganglia removed without injuring the sympathetic roots (ramus communicans albus et griseus), the common motor-

endings and the sensory endings disappear, but the fine non-medullated nerve-fibres with their small nerve-endings on the muscle-fibres remain unaltered. The sympathetic fibres for the muscles of the tongue seem to run, not in the hypoglossal, but in the chorda tympani (Boeke). Their endings on the muscle-fibres present the same aspect as the sympathetic endings in the muscles mentioned above. So the sympathetic innervation of the striated muscle-fibres seems to be put on a solid morphological basis.

Now these sympathetic nerve-endings have the same hypolemmal position as the common motor-endings, and indeed are often found embedded in the same mass of granular sarcoplasma. Moreover it is certainly of interest that surrounding the end-loops and end-nets of these sympathetic nerve-endings there may be distinguished exactly the same regular protoplasmic network as was described for the motor-endings. And indeed, where these sympathetic endings were found embedded in the sarcoplasm of the motor-ending itself, the same periterminal network was seen surrounding them both.

For smooth muscle-cells too, as I mentioned above, it was not only possible to show that the end-loops of the nerve-fibres enter the cell and lie embedded in the protoplasm, but here too they seemed to be in connection with a distinct reticulum.

As to the sensory nerve-endings, there are at least two different kinds of tactile end-organs which exhibit the same features. In the first place the corpuscles of Grandry in the skin covering the duck's bill (figs. 9, 10). As is known, these corpuscles consist of two (or three) large, flat, disc-like cells within a capsule, with the axis-cylinder terminating in a flattened expansion between the cells. In the numerous papers dealing with the histology of the corpuscles of Grandry the tactile disc is always regarded as being entirely independent of the two cells between which it is lying. But Gasiorowsky saw these cells degenerate after section of the nerve, and in a set of admirable Bielschowsky preparations of the skin of the duck's bill made by Dr. Heringa in my laboratory, not only the tactile disc could be seen in a syncytial connection with both cells, but inside the protoplasm of these cells there was visible a distinctly stained, sharply defined network in absolute continuous connection with the neurofibrillar structure of the tactile disc. It is seen extending throughout the whole thickness of the two adjoining cells, in short, it exhibits all the features so characteristic of the periterminal network surrounding the motor nerve-endings.

In the second place I have to mention the tactile discs in the epi-

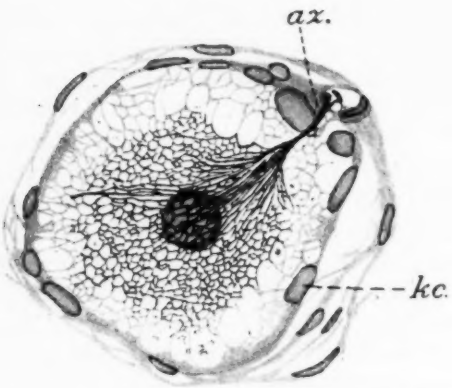

FIG. 9.—Tangential section of a corpuscle of Grandry of the skin of the duck's bill, with intraprotoplasmic network.

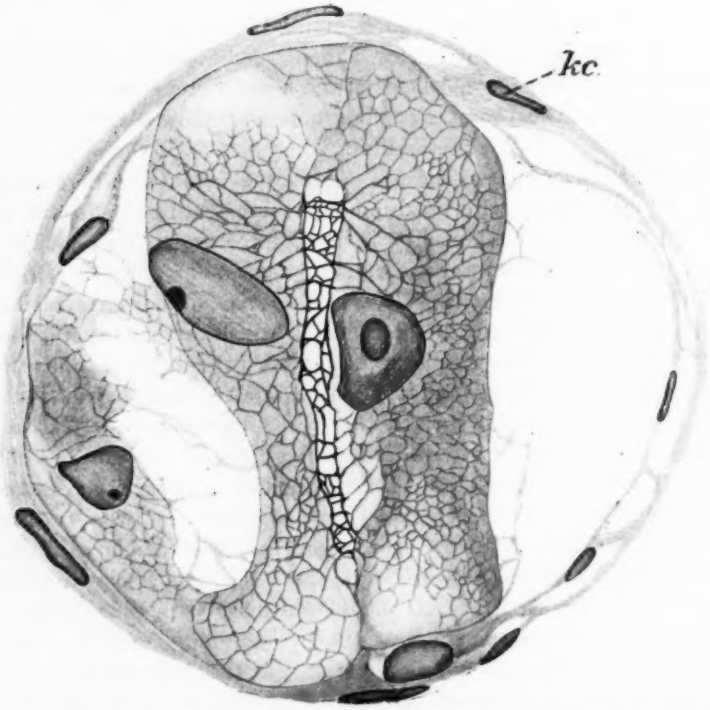

FIG. 10.—Cross-section of a corpuscle of Grandry of the skin of the duck's bill, with intraprotoplasmic network; *kc.* = capsule-cells.

dermis. It is generally assumed that these curious flattened or crescentic nervous expansions lie in the interstices of the deeper epithelium cells, for example, of the pig's snout, or in the organ of

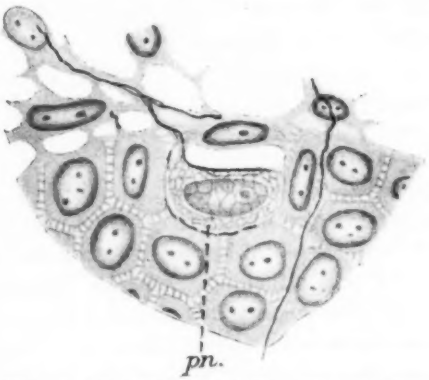

FIG. 11.—Cross-section of the epithelium of the organ of Eimer of a mole, with nerve-fibres entering the skin and a tactile cell with nerve-fibre and intraprotoplasmic network (*pn.*).

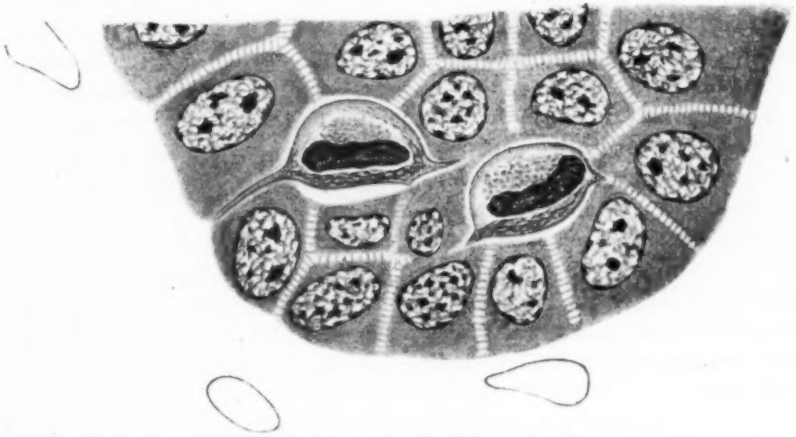

FIG. 12.—Transverse section through the epithelium of the skin of the pig's snout with two tactile cells of Merkel, showing the intraprotoplasmic position of the neurofibrillar network.

Eimer in the snout of the mole, and they may be shown to consist of a fine network of neurofibrillæ. From the earlier investigations of Merkel and Ranvier to recent times almost all writers on the subject agree on

this point, that they are lying between the cells of the epidermis. And yet, when we study them in very thin sections treated, fixed and stained with the utmost care, we come to the conclusion that they are not lying between the cells with a somewhat differentiated cell of the epithelium covering them, but that the neurofibrillar expansion, the tactile disc, lies inside the protoplasm of a differentiated cell of the epidermis. And, moreover, we see that the neurofibrillar reticulum is continued into a distinct protoplasmic network with fine regular meshes, extending throughout the cell (fig. 11) and exhibiting just the same features, characteristic of the periterminal network, as in the case of the corpuscles of Grandry.

These points, which can only be seen in the most carefully made preparations and with the use of the highest magnifying power, are not at all easy to study, and although I was able to make several observations on other sensory endings, as, for instance, the corpuscles of Herbst and the cells of the taste-buds, which seem to tend in the same direction, I do not think it advisable to give here the further details of these investigations.

But it seems to me that the observations recorded give us reason for supposing that not only in the case of the motor-endings but also in the different forms of the sensory nerve-endings there exists in the first place a close connection between the nerve-endings and the surrounding "lemmoblastic" elements that gives us a right to speak of a syncytial arrangement. And, secondly, we may assume that everywhere between the neurofibrillæ of these nerve-endings and the protoplasm is inserted a distinct reticulum, which seems to be the histological substratum for the receptive substance of the physiologists.

This may furnish a solid base for further study in this direction, and for the present-day histologist it is another proof of the inadequacy of the old cell theory, which regards the cells as independent self-sustaining units, and of the wonderful harmony in which all those units combine and act together to build up and sustain the higher organism, the individual.

THE RELATION OF THE HIND-BRAIN TO MICTURITION.[1]

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In a previous paper [1] experiments on the cat were described, from which, among others, the following conclusions were drawn:—

(1) There is a motor tone passing to the bladder through the pelvic nerves which arises in the central nervous system above the level of the lower thoracic region of the cord.

(2) Transection of the cord in the lower thoracic region abolishes reflex micturition, i.e., the reaction which ultimately occurs if a bladder is gradually artificially distended, leading to the expulsion of its contents.

(3) Transection of the cord in the lower thoracic region permanently abolishes micturition. The escape of urine which occurs when the stage of so-called spontaneous (or automatic) micturition has been reached differs from micturition in the essential respect that the bladder contraction and urethral relaxation are not co-ordinated, as is shown by the fact that the bladder is never emptied. When this escape of urine is provoked reflexly by perineal stimuli, the jets of urine passed can be seen to be synchronous with rhythmic contractions and relaxations of the perineal muscles.

The object of the present investigation was firstly to determine the level in the central nervous system of the origin of the motor tone of the bladder muscle. Secondly, to find out what parts of the central could be removed and micturition still take place. Thirdly, to find what simple reflexes are combined together to form micturition.

(1) Motor Tone of the Bladder Muscle.

The state of the bladder muscle was estimated, as in the previous experiments, by measuring its volume under a constant pressure of 10 cm. of water. The method, which was described before, consisted in cutting away the pubic arch, tying a brass catheter (size 10 Charrière)

[1] The expenses of this research were defrayed by the Graham Research Fund.

into the urethra just above the prostate in the male, and in the corresponding place in the female, and allowing all urine to escape from the bladder. The catheter was then connected with a closed graduated wash-bottle of water hung on pulleys in a bath which was kept warm and at a constant temperature within 3° C. The bottle was raised or lowered as the volume altered, except for the rhythmic undulations of contraction and relaxation which are usually present: 30 c.c. in the bottle caused a rise of pressure of just under 1 cm., hence as the undulations seldom produce a volume difference of more than 15 c.c., the maximum pressure variation would usually be about $\frac{1}{2}$ cm. of water. The volume was recorded by connecting the air space of the bottle with a Langley's piston recorder which was attached to a Keith Lucas' lever. As previously stated, the volume of urine secreted by cats under anæsthetics is negligible for the purposes of these experiments.

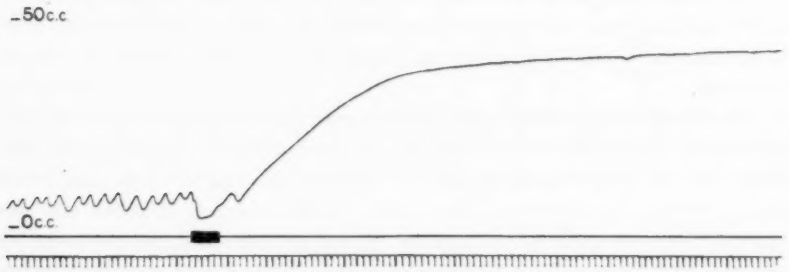

FIG. 1.—Cat, female. Ether. Splanchnic nerves divided. Bladder volume at 10 cm. H_2O pressure and 42° to 43° C. At the signal the spinal cord was divided between the first and second cervical roots. Time marker, five seconds.

A series of experiments was first made to find out if the same effect on the bladder volume was produced by dividing the cord in the upper cervical region as has been previously described in the case of the lower thoracic region [1], namely, an immediate abolition of any sharp undulatory contractions and a dilatation which persists, the dilatation being sometimes preceded by a sharp transient contraction. For this purpose the cats were anæsthetized with ether, a tracheotomy tube with two exits tied in the trachea, the catheter tied in the urethra as described, and dorsal part of the arch of the atlas cut away and the dura deep to it opened. Artificial respiration was then started and ether given with the respiration pump. After the volume had become reasonably constant, the cord was divided and the result on the volume noted. The effect was apparently exactly the same as in lower thoracic

transections and occurred with remarkable constancy (fig. 1); the only cases in which it appears not likely to occur are those with very large volumes and no undulatory contractions, a condition which is generally due to very deep anæsthesia or a bad general condition as a result of hæmorrhage. As in lower thoracic transections, division of the cord in the region of the first cervical root is followed by a persistent increase in volume and abolition of sharp rhythmic contractions, whether the hypogastric nerves are intact or divided. There are two factors in the upper cervical transection which might affect the bladder volume that do not occur in the lower thoracic region, namely, the fall of blood-pressure and the alteration in the amount of ether. That the effect is not due to the fall of blood-pressure was shown by experiments in which the cord was divided after previous division of the splanchnic nerves, the effect on the bladder volume being the same as when these nerves were intact. When the cats were having artificial respiration, as a rule, spontaneous breathing neither ceased nor was synchronous with the pump; they were, therefore, getting less ether before the spinal transection than after, when spontaneous respiration ceased, the ether regulator not having been altered: no steps were taken to find out what this difference was and, as will be shown later, ether both abolishes rhythmic undulations and causes a relaxation of the bladder in decerebrate cats: in this case, however, there is a very appreciable though not long interval before the effect occurs, whereas in the transection it is immediate (figs. 1 and 4, Part I), from which it seems that the effect cannot be due to any increase in the amount of ether taken after spontaneous respiration ceases. It was therefore concluded that the motor tone of the bladder arises in the brain.

A series of experiments was then made to determine whether the same effect on the bladder volume was produced by division of the mid-brain as by division of the cord. For this purpose the cats were tracheotomized and ether continued with an artificial respiration pump. A large part of the skull in front of the tentorium was removed and the dura opened. The brain section was made with a blunt knife, keeping it close against the tentorium. In some cases the brain in front of the section was removed in order to allow all hæmorrhage to come forwards and thereby avoid an increase of pressure in the posterior fossa. Two of the experiments were useless as death occurred a few minutes after the brain section. In the remaining five, one showed an abolition of undulatory contractions and a persistent increase in volume as is the case after division of the cord, one (fig. 2) showed an increase in volume

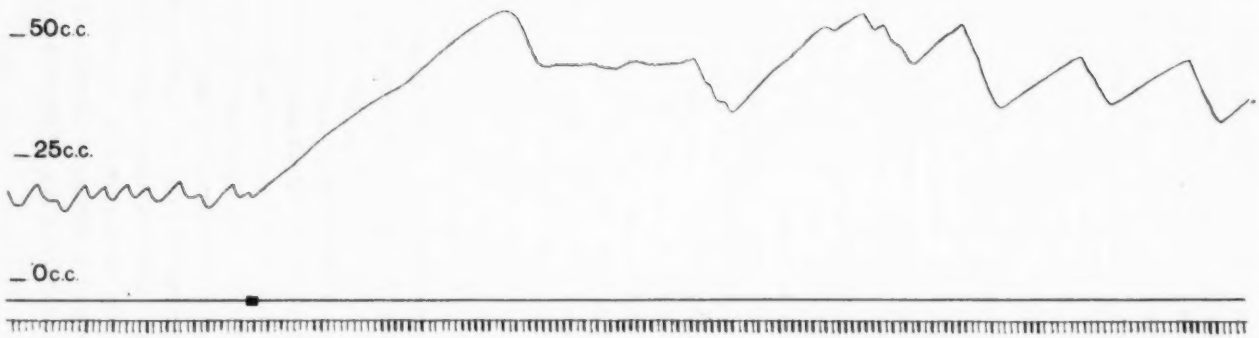

FIG. 2.—Cat, male. Ether. Bladder volume at 10 cm. H_2O pressure and 39° to 41° C. At the signal the mid-brain was divided through the superior colliculi and crura just in front of the superficial origins of the third nerves. Time marker, five seconds.

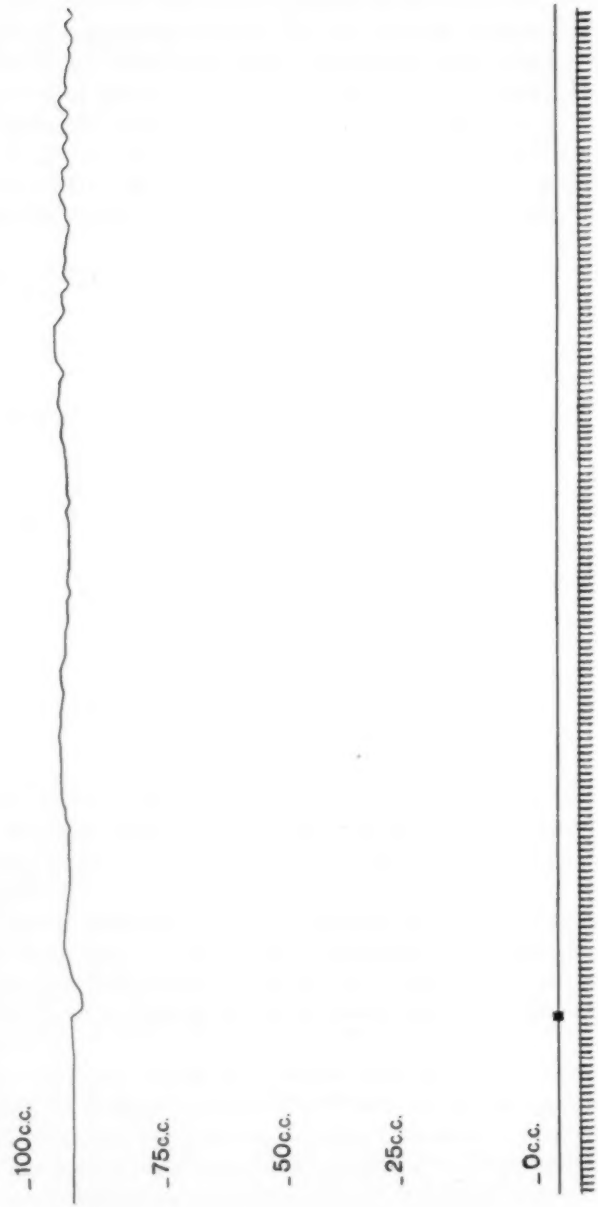

Fig. 3, Pt. I.

but large undulations became established again and the remaining three all showed diminution of volume with large undulations: in one of them (fig. 3, Parts I and II) the diminution took some time to begin, in the other two it was immediate. In the three experiments where a contraction followed the division of the mid-brain subsequent division of the upper cervical cord or lower part of the medulla was followed by the usual effects (fig. 3, Part III), namely, a persistent increase in volume and abolition of all large undulatory

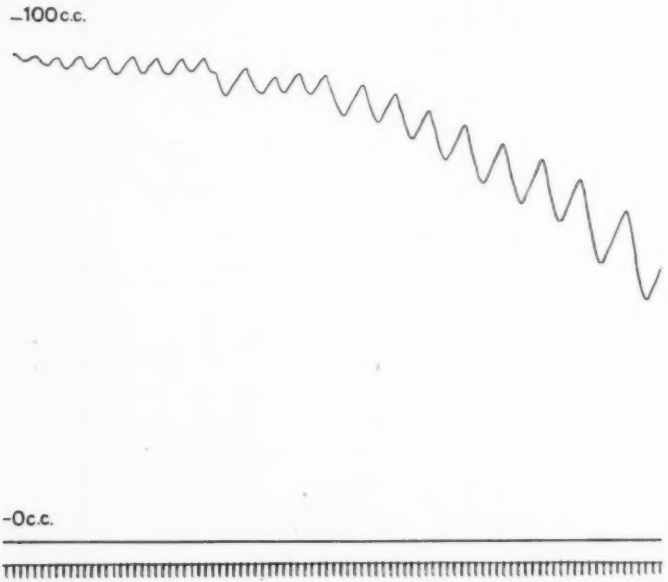

FIG. 3, Pt. II.

contractions. From these experiments it was concluded that the motor tone of the bladder arose somewhere between the lower part of the mid-brain and the lower part of the medulla. Subsequent experiments to localize its level of origin were made on decerebrate cats without anæsthetics.

Decerebration was performed under chloroform anæsthesia. The origin of the temporal muscle was detached from the skull. A trephine hole was made just in front of the position of the tentorium on the left side: this was enlarged with bone forceps and a large hole made in the dura. The division of the mid-brain was made as already stated and

the whole brain in front of it rapidly removed with the handle of a teaspoon. As stated by Bazett [2] the bleeding is very much less if the anæsthesia is very deep. The anæsthetic was stopped directly the the brain was removed. Bleeding was stopped by plugging the skull;

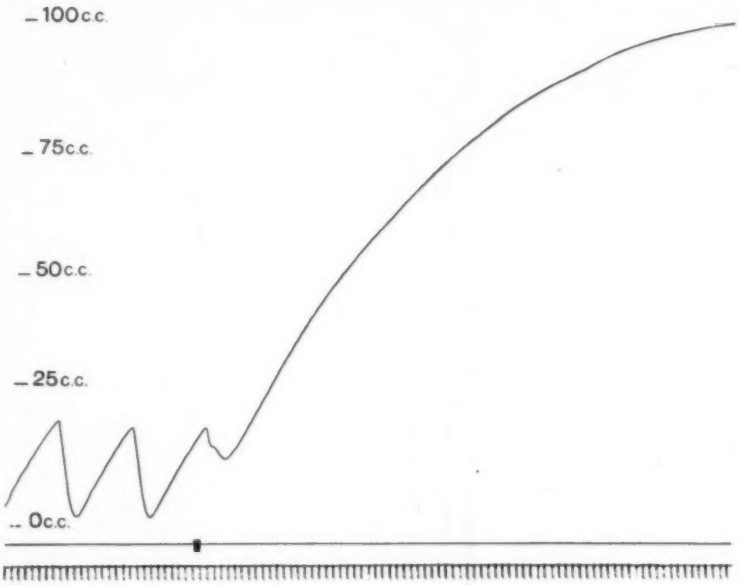

FIG. 3, Pt. III.

Cat. Female. Ether. Bladder volume at 10 cm. H_2O. At the signal in Part I the brain was divided just in front of the superior colliculi and the stalk of the pituitary.

Part II is ten seconds after Part I.

Part III is about six minutes after Part II. At the signal the cord was divided between the first and second cervical roots. The volume remained approximately the same till the animal was killed twenty minutes later. Time marked, five seconds.

In Parts I and III there was the same amount of air in the reservoir and piston recorder; the volumes represented in these two therefore correspond accurately. In Part II it was thought necessary to alter the amount of air in the reservoir to prevent the needle going off the tracing: after the experiment the 100 c.c. mark on this was made to correspond to that on Parts I and III: the other volumes of Part II do not, however, necessarily correspond accurately to those of Parts I and III, as with a Keith Lucas' lever a given change of volume does not produce the same excursion of the needle in all positions.

Horsley's method with a piece of freshly cut muscle placed over the sella turcica and plugged down with wool was found very effective. More than half an hour was always allowed to elapse before proceeding with the experiment. The volumes of decerebrate cats' bladders at

FIG. 4, Pt. I.

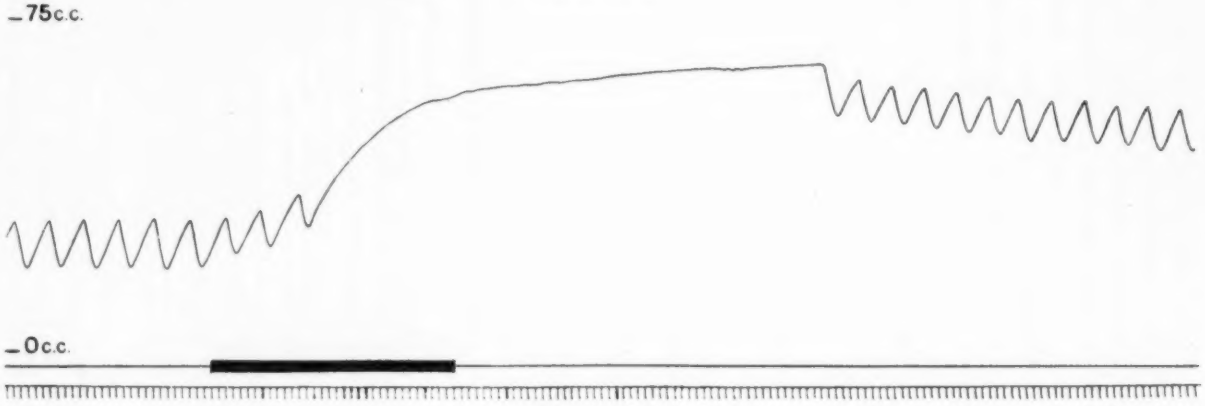

FIG. 4, Pt. II.

10 cm. of water pressure were on the whole much lower than those of intact cats under ether.

Before proceeding further with experiments to determine the level in the brain at which the motor tone of the bladder arises it was thought advisable to find out the effect of curare and also of ether and chloroform on the bladder volume of decerebrate cats. Chloroform and ether were given by putting one exit of a three-way tracheotomy tube into the air space of a Wolff's bottle containing some of the corresponding liquid, the second exit being clamped to a greater or lesser degree: by this means rather concentrated vapour was given. In the three experiments which were made the effect was the same: the volume gradually increased, in about a minute the undulatory contractions ceased and the

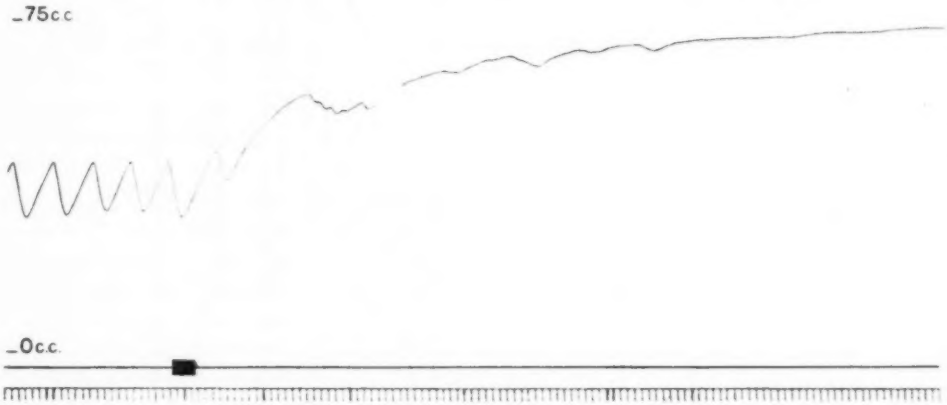

FIG. 4, Pt. III.

volume then increased more rapidly (fig. 4). It is the same effect as that of dividing the cord but it is not produced as suddenly. To show the effects of curare on the bladder volume, a solution of it, made in slightly acidified Ringer's solution, was injected into the external jugular vein. Very small doses (e.g., 1 c.c. of $\frac{1}{4}$ per cent.) produced an increase in the bladder volume without abolishing the undulatory contractions. When larger doses were given (fig. 4, Part III) these were abolished as well and the immediate effect then exactly resembled that of dividing the cord. This effect could be obtained with doses insufficient to abolish the corneal reflex. When the animals lived long enough there was a tendency for the volume to recover to some extent and for the undulatory contractions to reappear (fig. 4, Part IV). The

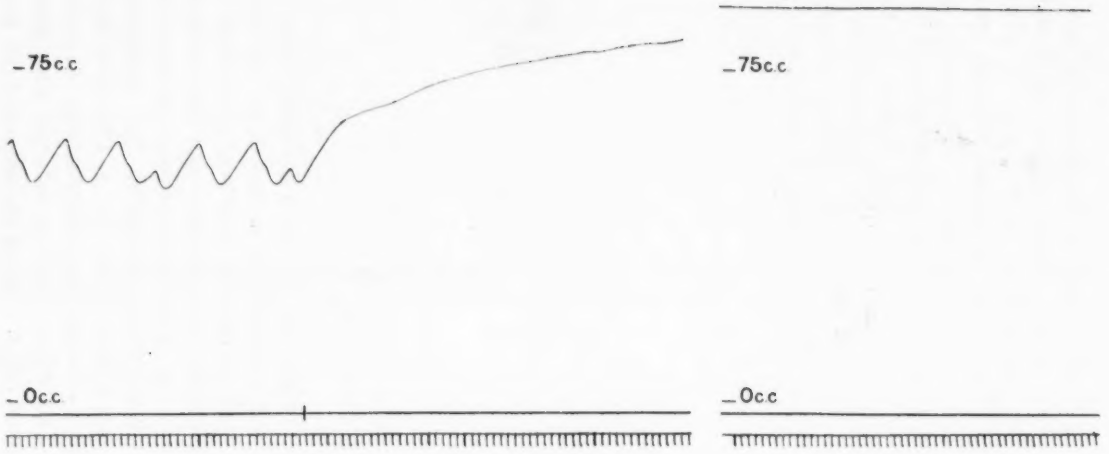

FIG. 4, Pts. IV and V.

Cat, male ; decerebrate. Bladder volume at 10 cm. H_2O pressure. Time marker, five seconds.

Part I : The fore-brain had been removed and the anæsthetic stopped two hours and twenty minutes before the beginning of the tracing. During the signal ether was given ; the corneal reflex was absent two minutes after the ether was started.

Part II : Commencing twenty-nine minutes after the end of Part I. During the signal chloroform was given, the corneal reflex was absent after two minutes, and after three, when the chloroform was stopped, respiration ceased ; artificial respiration was then performed for the next three minutes.

Part III : Fifty-five minutes after Part II. Artificial respiration : 2 c.c. of 2 per cent. curare were given subcutaneously fifteen to sixteen minutes before without obvious effect. During the signal 2 c.c. of 2 per cent. curare were given into the external jugular vein.

Part IV : Forty-eight minutes after Part III. At the signal the medulla was divided between the superficial origins of the hypoglossal and first cervical nerves.

Part V : One hour fifteen minutes after Part IV.

final volume was, however, considerably greater than the original one.

Curare was used in the series of seven experiments made to determine the level at which the motor tone of the bladder arises. The object of its use was to eliminate the disturbances in the bladder volume due to contractions of the abdominal muscles occurring in the powerful movements which always occur in a "spinal" cat. It is most important to know whether the volume alteration commences at the moment of division, in which case it is most likely directly due to the division, or a short time after, in which case it may be a secondary result produced by hæmorrhage: if curare is not used it is at this particular time that the muscular contractions are strongest and most frequent. The curare was given subcutaneously in doses of about $1\frac{1}{4}$ c.c. of 1 per cent. solution per kilo; more than an hour was allowed to elapse before proceeding with the experiment. Subcutaneously curare seemed less toxic and to produce its own characteristic effects just as well. The bladder volumes, in experiments where it had been given, seemed on the whole greater than in those where it had not, as would be expected from the result following its injection intravenously. In three of the seven experiments sections were made of the pons in its upper half; in all the volume recovered and undulations started again in two and were not abolished in the third; in two of these the section was not quite anatomically complete. The third was the lowest section; it went just in front of the superficial origins of the fifth nerves below and through the posterior parts of the inferior colliculi above. In all the seven experiments sections were made which produced a persistent dilatation which failed to pass away; the levels of these were 4 mm. below the upper border of the medulla, through the origins of the sixth nerves (two experiments), between the medulla and pons, through the lowest part of the pons (two experiments), and through the middle of the pons (fig. 5). Therefore, the highest section which gave the result and the lowest one which did not were both about the middle of the pons. From this it can be concluded that the tonic influence the central nervous system exercises on the bladder through the pelvic nerves arises in the central nervous system at about the level of the middle of the pons.

(2) Reflex Micturition.

A series of eighteen experiments was performed in the following way on female cats. The fore-brain was removed under chloroform as previously described and the chloroform administration stopped immedi-

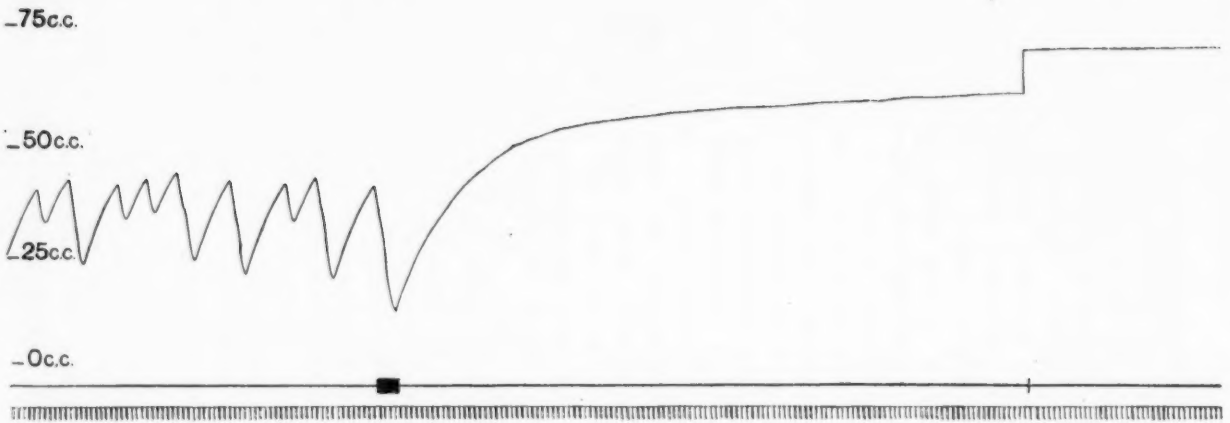

FIG. 5.—Cat, male. Bladder volume at 10 cm. H_2O pressure. The brain had been divided through the inferior colliculi, chloroform stopped, and the brain in front of the division removed two hours three minutes before the beginning of the tracing; 3 c.c. of 1 per cent. curare were given subcutaneously twenty-eight minutes after removal of the brain. At the first signal the brain was divided through the posterior part of the inferior colliculi above and the middle of the pons below. At the second signal the drum was stopped twenty minutes. Time marker, five seconds.

ately after. More than half an hour was then allowed to elapse before proceeding with the experiment. The cat was placed on its side at the end of a warm box inclined at about 15° to the horizontal, so that any fluid passed by the urethra ran on to a gutter at the end of a box into a receiver underneath, from which it could be measured. A brass catheter (size 7 Charrière) was then passed and the bladder emptied of any urine it might contain. In the earlier experiments a median incision was made dorsalwards into the perineal body to facilitate passing the catheter; this was afterwards abandoned as it was found unnecessary and also because it was suspected that the reflex might be interfered with by the irritation necessarily set up. The catheter was then connected, by means of a rubber tube, fitted with a Mohr's clip, to a warm water reservoir about 50 cm. above it; a metal worm in a warm water bath was interposed between the reservoir and the catheter to eliminate some of the cooling certain to occur in the tubes between the two. To make an observation the clip was removed from the rubber connecting tube and the vulval opening watched; when a definite escape of water at the side of the urethra had been established, e.g., after the escape of four to six drops, the tube was clamped off and the catheter removed from the urethra, the quantity of water passed was measured and the catheter passed again after to determine the quantity remaining in the bladder, if any. This was repeated several times in each experiment.

It was found that the water was passed in a continuous stream at first and often finished with a short series of jets; immediately after the passage of water had stopped one or more strong contractions of the perineal muscles usually occurred. The commencement was often initiated by an abdominal contraction of sufficient force to move the limbs and arch the back; this sometimes occurred just before and sometimes just after the withdrawal of the catheter. After the first observation had been made the volume necessary to provoke the reaction was more or less the same in each individual in subsequent observations. It was nearly always less than the volume of urine passed at a normal micturition and often very many times less, for instance, in seven out of the eighteen experiments it was constantly less than 20 c.c. The rapidity of filling the bladder, the fact that, though warm, the water was probably never exactly at the body temperature, and possibly also the presence of a catheter in the urethra, obviously may be sufficient to account for this deviation from the normal without the supposition that with the removal of the fore-brain there has been a removal of inhibition,

though as will be shown later there is a certain amount of reason to believe this also occurs. In all observations of eleven out of the eighteen experiments the amount of water left in the bladder at the end of the act was either none at all or less than 1 c.c. In three others it was 1½ to 3 c.c., in the first observation in each, and less than 1 c.c. in the subsequent observations in each. In one other it was 1½ to 2½ c.c. in each observation. In these fifteen experiments therefore the micturition reflex was as nearly perfect as it is likely to be found. The remaining three all had very appreciable quantities remaining in the bladder at the end; an incision had been made into the perineum in all three cases; all three showed very strong abdominal contractions at the commencement of the act and the initial stream of water had an explosive force but stopped quite suddenly as if the urethral muscles had closed too soon. It is possible that the perineal incision may have had something to do with the unusual phenomena seen in these three experiments; it was made in eight of the other fifteen experiments, so this does not seem very probable. It seems much more likely that pressure on the remaining part of the brain from hæmorrhage into the posterior fossa of the skull was the cause, since the same effect was sometimes seen, in experiments where the passage of water had previously been normal, after interference with the posterior fossa or its contents. In one of the three experiments 5 to 6 c.c. were passed at each observation and the quantity left in the bladder gradually rose, being 11 c.c., 23 c.c., and 41 c.c.; during the fourth observation the cat developed spasmodic inspirations, scratch movements followed and then respiration ceased, while the heart continued beating; a thin clot was found on the ventral surface of the medulla and pons; it must be admitted however that this can sometimes be found in cats after decerebration in the way described which have not had any symptoms of pressure in the posterior fossa. In the second 4 c.c. to 8 c.c. of water were passed at each observation and the water remaining in the bladder was 17 c.c., 12 c.c. and 8 c.c. respectively: the cat died less than half an hour after the third observation and clots were present as in the last one. In the third experiment the residual water was relatively less and diminished with time, till in the last observation 62 c.c. were passed and 10 c.c. remained in the bladder; the cat died soon after this but the cause of death was uncertain as respiration stopped after some spasmodic inspirations five minutes after the injection of 5 c.c. of 10 per cent. cocaine hydrochloride into the bladder, an amount subsequently found sufficient to produce at any rate very severe symptoms. Whatever

the cause of these three exceptions the facts stated seemed quite sufficient to justify the conclusion that reflex micturition takes place normally in cats in the absence of the whole of the fore-brain and anterior part of the mid-brain.

Chloroform seems to have a very depressing effect on reflex micturition even when the anæsthesia is light. In eight of the above experiments an observation was made while the cat was under chloroform and before the fore-brain had been removed. In all the eight a very much larger quantity of water had entered the bladder before it escaped from the urethra than occurred after decerebration when the anæsthetic had been stopped some time. In all the eight a very considerable amount remained in the bladder after the escape from the urethra had stopped; in six this was more than twice the amount passed. Instead of being passed in a forcible stream the water came away in drops in these experiments, which were sometimes fast enough to form a continuous stream at the very beginning but never later. In another experiment, after decerebration, four observations were made and 8 c.c. passed in each one with a residual of nothing to five drops: chloroform was then given and the observation repeated, 7 c.c. were passed and 78 c.c. remained in the bladder.

In six of the experiments observations were made after injecting cocaine hydrochloride solution into the bladder. The solutions were left in fifteen minutes or more. As already stated, general symptoms are easily produced by this with strong solutions (e.g., 5 c.c. of 10 per cent.). The symptoms are a marked increase in respirations and an increase in the rigidity, particularly as shown by head retraction.

Five c.c. of 1 per cent. solution in one experiment had very little, if any, effect. In the remaining five intermediate strengths or quantities were used; they all required a greatly increased volume of water to provoke an escape of it from the urethra, and all had a very considerable volume left in the bladder after the passage of water had stopped. These results agree with those of Shattock [10] with cats under A.C.E. mixture. In all cases the effects were very transitory.

In a few cases the bladders were examined with X-ray plates after filling them with 10 per cent. colloid silver or a suspension of bismuth carbonate. In no case was a shadow present between the internal urinary meatus and the compressor urethræ unless the solution had actually been passed while the plate was being taken, and this was so whether the bladder had previously been injected with cocaine or not. Hence the solution is held at the internal meatus in decerebrate cats,

and not, as in those which have had a transection of the cord, at the compressor urethræ.

In two of the experiments the cord was divided at its union with the medulla and in one the medulla at the apex of the calamus. Observations made after showed the following changes. Firstly, the water did not escape from the urethra till the bladder contained a greater volume than that which was necessary to provoke it previously. Secondly, the amount passed after withdrawal of the catheter was smaller and instead of being passed in a forcible stream came away in a series of drops which sometimes showed rhythmic accelerations corresponding with visible rhythmic relaxations and contractions of the perineal muscles. Thirdly, when strong perineal contractions at the end of the act had been present before the division, they were absent after. Fourthly, a large amount of water remained in the bladder after the passage from the urethra had stopped. Reflex micturition was therefore abolished in these experiments, from which it may be concluded that it depends on the integrity of some part of the brain between the apex of the calamus and the mid-brain and on the connection of this part with the central nervous system below it. Reflex micturition had been shown by Elliott [4] to be abolished by destruction of the brain; he suggested that this effect was due to shock. Other reflexes, however, do not seem to be similarly affected; the shock can hardly be greater than that of removal of the fore-brain, and further, as stated in a previous paper [1], it could not be obtained some time (five weeks) after the cord had been divided. In three of the experiments the cerebellum was removed; post mortem it was found in all that the removal was incomplete, though the fragments remaining were small. In one of these experiments there were no changes in the reflex micturition after the removal, except that the bladder only tolerated about half the distension it did before. In the second the water was passed in a series of forcible jets instead of in a stream in the earlier observations after removal of the cerebellum, and in the later ones in drops only; in the earlier observations little or no water was left in the bladder at the end, while in the later ones there was a very considerable quantity. In the last the water was passed in a very forcible stream accompanied by a strong contraction of the abdominal muscles; the stream usually stopped suddenly and there was always a large amount of water left in the bladder; this cat developed spasmodic inspirations about an hour and twenty minutes after removal of the cerebellum. It seems probable, therefore, that removal of the cerebellum has no effect

on reflex micturition and that the effects of the operation, when any are present, are due to other parts of the brain below the tentorium being affected by hæmorrhage. In another experiment an incomplete division at the level of the upper part of the pons was made; spasmodic inspirations developed thirty-six minutes later, and reflex micturition during this time showed the changes described in the last two experiments in which the cerebellum had been removed.

(3) The Reflexes composing Reflex Micturition.

The first fifteen experiments made to analyse reflex micturition into its component parts were performed as follows: Decerebrate female cats which had come round from the chloroform were used and a catheter passed as in the experiments on reflex micturition. The bladder was distended from a burette of warm water, so that known amounts in known times could be run in. Interposed between the burette and the catheter were: (1) a metal worm in a warm-water bath next the catheter; and (2) a vertical tube, serving as a water manometer next the burette. Eighty cm. of the manometer tube contained 1 c.c., so pressure alterations produced no appreciable volume alteration. The bladder pressure could, of course, only be read when the clip of the burette was shut.

In these experiments it was found that active contraction of the bladder could be produced in one of two ways, by distension of the bladder to a given volume or by slightly moving the catheter in and out in the direction of the urethra.

To investigate the contraction resulting from distension, equal volumes of water, usually $2\frac{1}{2}$ to 10 c.c., were run into the bladder at definite intervals of time, usually every other minute, and the pressures noted at the end of one minute after each increase of volume. When a certain volume was arrived at it was found that the bladder pressure rose with or without an abdominal contraction, and this was usually accompanied by an escape of the water from the bladder at the side of the catheter; the catheter was then disconnected to empty any bladder contents that might not have escaped, and the same series of operations repeated several times. The volumes tolerated by individual cats varied considerably, but after the first reaction had been produced the subsequent ones in the same cat occurred at more or less the same volumes, provided the distension had been made at the same rate. The increases of pressure produced by increases of volume within the limits of toleration were not great, but when that limit was reached the

pressure would at once, or in a few seconds, rise to three to fifteen times its previous value and gradually fall with the escape of the water from the urethra. It was noticed that the greatest height to which the pressure rose could be seen, at any rate often, to occur after the escape of the water had become well established; this is probably accounted for by the next reflex to be described. In some cases the maximum rise of pressure was preceded by one or more rises, each one larger than the one before; this may have been due to the catheter causing obstruction to the urethra, so that the first increase was incompetent to start micturition. There can be no doubt about the increase of pressure seen in these experiments being due to an active contraction for the following reasons. Firstly, though in the case of a non-living elastic bag a given increment of volume may give a larger pressure increase if it is applied when the bag is tensely distended than if it is applied when the bag is lax, it could not give an increase of 200 to 1,400 per cent. when the last equal increment of volume had given rise to hardly any pressure increase. Secondly, though in most cases the reaction naturally occurred when the water was actually being run in, in quite a large number it did not occur till after the clip of the burette was shut, and then the whole rise of 200 to 1,400 per cent. was actually seen. Thirdly, in the case of a non-living elastic bag, however great the pressure increase resulting from an increase of volume, it cannot exceed that at which the fluid is run in, whereas in the present experiments when the reactions occurred while the fluid was being run in, it often happened that the monometer pressure was greater than that in the burette, and water flowed back into the latter. This reflex contraction of the bladder resulting from distension occurred in four experiments where curare had been given which eliminated all abdominal contraction; the pressure rise was not quite as marked in these experiments, and there was always an appreciable volume of water remaining in the bladder after the reaction had finished, which is what would be expected from what has already been stated about the effect of curare on the motor tone of the bladder. In one experiment the effect was obtained after division of the brain through the upper part of the pons. In five experiments the medulla was divided at various levels between its upper border and the apex of the calamus; the effect was the same in all. The distension being carried on at the same rate as before making the brain section, it was found that the pressure given by a volume just below that which previously produced the reaction was approximately the same as that given by the same

volume before the brain section. When the distension was increased, it was found that the bladder tolerated a greater volume than before, and that the pressure rose gradually with each volume increment till escape of water from the urethra finally occurred; after a small amount had escaped the escape stopped, the pressure dropping slightly but remaining higher than it had been at any time before the brain section was made apart from the rise with the reaction. After the brain section no rise of pressure as high as the reactionary one could be obtained by slow increments of volume, because whenever the pressure rose above a certain amount escape started from the urethra, tending to bring it down again; at the same time a higher pressure could be maintained after the section than before, since the corresponding volume would have evoked the reaction before the section and caused emptying of the bladder. In eight of the fifteen experiments cocaine was injected into the bladder; the effect of this on the bladder was exactly the same as that of dividing the medulla; the bladder tolerated a greater volume than before, and no sign of a reactionary contraction was observed. After both cocaine and division of the medulla rhythmic alterations in pressure were sometimes seen over and above those due to respiration; these had no resemblance to the reactionary contraction with gradually increasing distension, because firstly they were much less in degree; secondly, they were more or less equal and quite regular for the same volume; and lastly, they usually diminished and finally disappeared as the volume was increased up to that giving a pressure which caused escape from the urethra; in no case were they increased by increasing the volume to this degree. In three experiments cocaine was injected into the bladder after division of the medulla; it then had no effect either on the final volume tolerated or on the pressures corresponding to the various volumes retained. These results differ from those of Shattock [10], who found the intravesical injection of cocaine was followed by an increased toleration to distension both in pithed cats and in those under A.C.E.

The second way in which active contraction of the bladder was evoked was by withdrawing the catheter a fraction of a centimetre and then pushing it back to its original position; the stimulus of a smooth foreign body moving in the urethra would appear to be the same as a stream of urine passing through it. It was found that in intolerant bladders of low capacity an increase of pressure followed this with any degree of distension, but in bladders of larger capacity a certain degree of distension was necessary for its production and

that the greater the distension the greater the rise of pressure evoked by this form of stimulus. The rise of pressure produced in this way was on the whole not quite as large as that produced by distension, the largest seen being thirteen and a half times the original; in many cases, it was, however, quite enough to start reflex micturition, the bladder contents being passed at the side of the catheter: as in the first reflex, this one can be obtained after curare and after division of both hypogastric nerves. In three of the experiments the pudic nerves were divided. In one the reflex did not appear affected by the operation. In the second it was certainly more difficult to elicit and when the rise of pressure did occur it was accompanied by a strong abdominal contraction. In the third the hypogastric nerves had previously been divided; a rise of pressure still occurred on moving the catheter after division of the pudic nerves, but it was so small as to raise doubts as to whether it was due to contraction of the bladder. After division of the medulla either no increase in pressure at all followed the movement of the catheter or it was small enough to be considered as due to the mechanical effect and not to bladder contraction at all. Injection of cocaine into the bladder abolished the reflex in some cases and not in others; anæsthesia of parts of the urethra by this means is probably fortuitous, so if the reflex arises from part of the urethra this is what would be expected. From these results it was concluded that in micturition there are two reflexes leading to a powerful contraction of the bladder; one arises from distension of the bladder and the other from urine running through the urethra; both reflexes are destroyed by transection of the medulla.

For the purpose for which it was used the method just described is open to three objections. Firstly, as the results themselves show, at any rate under certain conditions, the presence of a catheter in the urethra is a disturbing factor. Secondly, the movement in and out of the catheter itself may produce a pressure alteration which is unknown. Thirdly, though it is improbable, the second reflex contraction described might be due to the end of the catheter touching the wall of the bladder and not to the catheter stimulating the walls of the urethra. For these reasons and to examine the efferent side of the urethral reflexes another method was used.

An examination of the distribution of the branches of the hypogastric plexus to the bladder and urethra showed that it appeared anatomically possible to divide the bladder at its neck, i.e., just

proximal to where the urethra begins to expand, and tie a cannula into both cut ends so that the pressure of urethral resistance and of bladder distension could be observed simultaneously.

The method consisted in making a median suprapubic incision, and exposing the ventral surface of the neck of the bladder. A ligature on a curved needle was then put round it without dissecting its sides or dorsal surface and taking care to keep the needle close against the bladder, an incision was made into the neck distal to the ligature and a glass cannula tied in. A second thread was then passed in the same way distal to the incision in the bladder neck and a second cannula, which except in the earlier experiments was filled with water, tied into the cut end continuous with the urethra. The part of the circumference of the bladder neck not already divided was then cut through so that the two parts were completely separated. A metal worm in a warm-water bath was interposed between each cannula and its manometer. The water manometers used were vertical tubes with a T-piece at the lower ends fitted with clamps; they could be filled by connecting a burette to the ends of the T-pieces not connected with the cannulæ. The tube of the urethral manometer was wide (1 c.c. = 3 cm.), so that an escape of water from the external meatus should not cause a very rapid fall of pressure. The tube of the bladder manometer was narrow (1 c.c. = 34 cm.), so that a large pressure alteration should not cause a very marked volume alteration. Cats of both sexes were used, but females were found more satisfactory, partly, no doubt, because the anterior part of the urethra which is useless for micturition is absent, and probably partly because the urethral resistance at the compressor is less than in the male.

Five reflexes were found by this method, three leading to contraction of the bladder and two leading to relaxation of the urethra. The three leading to contraction of the bladder were those already described and will be considered first.

The reflex contraction of the bladder provoked by its own distension was examined in the same way as in the previous series of experiments and the results obtained were similar. The bladder was distended by running in equal volumes of water (2½ to 10 c.c.) every other minute and the pressure noted a minute after each increase of volume. The volumes at which reaction occurred were about the same for each individual, provided the rate of distension was the same, but varied considerably in different individuals. The volume just below that

which provoked the reaction gave an intravesical pressure which was always less than 10 cm. and sometimes less than 5 cm.; the height of the reactionary contraction gave a pressure from 40 to over 90 cm., the highest pressure which the manometer used would record. The contraction usually took place while the water was actually being run in, but not infrequently it took place a few seconds after the volume increase had finished and then its whole extent could be actually seen. It is not implied from this that the stimulus is a factor of the volume, it is clearly a factor of the intravesical pressure and not volume, since a reaction can be provoked by a much smaller volume if it is run into the bladder fast than by that which is necessary if run into it slowly: the same conclusion was arrived at by Mosso and Pellacani [9] in an entirely different way and as a clinical observation it is probably much older still. The few experiments which deviated from this description were clearly due to interference, probably by hæmorrhage, with the brain in the posterior fossa and will be described after the conditions which destroy the reflex have been considered. The reflex can be obtained after division of both pudic nerves or both hypogastric nerves: both afferent and efferent paths are therefore by exclusion in the pelvic nerves as was originally shown by Guyon [8]. The reflex was not obtained if the pelvic nerves were divided, if the spinal cord was divided either in the lower thoracic or the upper cervical region, or if the interior of the bladder was cocainized. The bladder on distension in all three cases behaved in the same way, the only reservation to be made being that in the case of cocaine the complete effect only lasts a few minutes; it is therefore necessary to run in such volumes every minute that the whole observation may be completed quickly after the cocaine has been let out of the bladder. The pressures corresponding to volumes tolerated before any of these three operations were performed were not less for the same volumes after performance of the operation. When the bladder was distended further, the pressure increased with each volume increment, the increases of pressure becoming greater for each volume increment the greater the distension of the bladder. No sign of a reactionary contraction was ever seen and the only thing that could possibly be mistaken for it was the presence in some experiments of the undulatory contractions already described; in addition to the reasons already given showing that these are not reflex contractions they were found to be present in some cases after division of both pelvic nerves. The statement that the pressures given by volumes tolerated before division of the cord or pelvic nerves are not less for the

same volumes after may at first sight appear at variance with the results already described and tracings shown in the section on the motor tone of the bladder. These experiments were, however, made at a pressure of 10 cm. and as already stated the bladder of a decerebrate cat reacts before this pressure is arrived at: the bladders in the volume experiments were therefore probably in a continual state of reaction and the sharp undulations shown in the curves really represent reactionary contractions. This conclusion is confirmed by the fact, already stated, that the most marked volume effects following these operations were found to occur when marked sharp undulatory contractions were present. From these results it is concluded that the reflex contraction of the bladder resulting from distension has both its paths in the pelvic nerves and that the paths down the central nervous system from the level of origin of the motor tone of the bladder to the origin of the sacral roots forming the pelvic nerves are essential to its existence. The experiments mentioned, where the behaviour of the bladder differed from that described, either showed the same volume and pressure relations as if the cord had been divided or with low volumes powerful irregular contractions were present as shown by considerable irregular increases of pressure; as the volume was increased these contractions usually got greater, but from the fact that the pressure they started from was so irregular it was impossible to say that any one of them was a reactionary contraction. Both these conditions appear to be due to failure of the functions of the lower part of the brain, the second being the early stage and the first the final one. The reasons for concluding this are, firstly, that in some experiments the bladder first reacted in the usual way, later the powerful irregular contractions set in, and finally it behaved as if the cord had been divided; some time after this respiration ceased while the heart continued beating, this having been preceded by spasmodic inspirations and these by rapid respiration with an occasional deep inspiration. Secondly, in those experiments where all stages were not seen but where the abnormality occurred, respiratory failure developed in the same way later. From the fact that the bladder motor tone centre is higher than the respiratory centre, the bladder would be expected to be affected before the respiration in decerebrate animals.

The second reflex leading to a powerful contraction of the bladder was evoked by running water through the urethra. The rise of pressure so produced was about the same degree as that in the last reflex described. It was investigated by distending the bladder to a volume

below that which had previously been found to produce a reflex contraction and after waiting a few minutes for the immediate effect of distension to pass off the pressure was read and noted at short intervals. The urethral stimulus was applied by gradually increasing the pressure in the urethral manometer till it became just sufficient to overcome its resistance, water would then escape from the external meatus and the pressure in the urethral manometer would fall in consequence. After an appreciable latent period of about two seconds the bladder pressure began to rise, in several experiments it rose to over 90 cm. The pressure remained up, though not at its highest, until the escape of water from the external meatus stopped. The contraction of the bladder occurred in these circumstances even when it was practically empty, since when the bladder cannula was disconnected from its manometer a few drops of water could at times be seen to be expelled from its open end after the escape of water from the external meatus had started. This reflex was not affected by division of both hypogastric nerves. It could be obtained after cocainizing the interior of the bladder, showing that the bladder motility is not impaired by cocaine and therefore that the abolition of the reflex contraction resulting from distension described in the last paragraph must be due to sensory loss. The reflex was abolished by division of both pelvic nerves, or division of the spinal cord either in the lower thoracic or upper cervical regions. From these facts it may be concluded that the efferent path of this reflex in the pelvic nerves and, as in the first reflex, that the paths from the middle of the pons for the motor tone of the bladder must be intact for its existence. In nine experiments the pudic nerves were divided after this reflex had been obtained, the pelvic and hypogastric nerves being intact: in eight the reflex could not be obtained after, but in the remaining one it could; in this experiment it was abolished by subsequent division of the hypogastric nerves. Afferent fibres for this reflex must therefore be constantly present in the pudic nerves: whether these are only exceptionally present in the hypogastric or whether they are constantly present and were usually destroyed by the operative precedures on the neck of the bladder was not decided.

In making the experiments just described a reflex rise of intravesical pressure was observed which was easily distinguished from that just described and which was first believed to be due to some extravesical cause. When the pressure in the urethral manometer was being raised, as the pressure equal to the urethral resistance was

approached sharp transitory rises of intravesical pressure amounting only to a few centimetres were very frequently observed. The stimulus of this reflex was evidently distension of the posterior urethra, since in one experiment as the urethral pressure was gradually raised it fell ½ cm. each time it reached 30 cm., though there was no escape of water from the meatus till it was raised to 50 cm.: when it fell this ½ cm. the bladder pressure immediately gave a small transitory rise and such rises continued after. This reflex rise of intravesical pressure was, however, seen to its greatest extent if instead of gradually increasing the urethral pressure this was suddenly increased by removing the clamp when the pressure in the manometer was just above that previously found necessary to force the sphincter. In these circumstances water did not usually escape from the external meatus for some seconds, but immediately the urethral pressure was increased the bladder pressure rose, often 10 cm., and rapidly fell again at once, the rise and fall taking about five seconds. That this rise of pressure was due to a bladder contraction was demonstrated by exposing the bladder through a lumbar incision and seeing it contract. This reflex is present after division of the spinal cord, both pelvic nerves, or both pudic nerves, but is abolished by division of both hypogastric nerves or the lumbar splanchnic nerves. Both paths of the reflex are therefore in the hypogastric nerves.

The fourth and fifth reflexes found by this method lead to relaxation of the urethra; they were evoked respectively by running water through the urethra and by distending the bladder. The fourth reflex will be considered first.

If the bladder was empty, or only contained a volume less than that which caused a reflex contraction, and the urethral manometer was gradually filled by running in equal quantities of water at equal intervals of time, when a certain pressure was reached water escaped from the urethra, usually after a few preliminary contractions of the perineal muscles. The escape was rapid and continuous and was either uniform or showed rhythmic accelerations, but, at any rate in females, never stopped and afterwards began again. As a result of the escape of the water the pressure fell and, when the escape had stopped, remained constant at a considerably lower value. If now the same operation was repeated the escape from the meatus did not start again till approximately the same pressure as that which produced it previously was arrived at, when the escape started and the pressure fell to approximately the same value as before. The figures from the first

experiment of this kind made (on a female cat) may be quoted for the sake of clearness. The pressure was gradually raised till escape of water from the meatus started when it was 34 cm., in the next half minute it fell to 10½ cm., when the escape stopped and in consequence the pressure remained constant; the pressure was then increased again by 5 cm. each minute, escape of water from the meatus started again when the pressure was between 30 and 35 cm., and in the next half minute fell to 11 cm., when the escape stopped and the pressure remained constant. The fact that pressures above 11 cm. and below 30 cm. were resisted by the urethral muscles when the water was not escaping from the meatus, and not when it was, can only be due to a reflex relaxation of the urethra while the water is flowing. This reflex is still obtainable after division of both hypogastric nerves. After division of both pudic nerves water escapes from the external meatus under a much lower pressure than previously, unless the pressure is very high the escape is slow; when finally the escape has stopped any increase of pressure, even 1 cm., causes the escape to start again at once. The reflex is therefore abolished by division of both pudic nerves. It is a curious fact that sometimes the pressure to which the urethral manometer fell at the end of the reflex was lower than that of the urethral resistance after division of both pudic nerves. In this connection it is well to remember that the pelvic nerves are known to send inhibitory and the hypogastric motor fibres to the smooth muscle of the urethra [11], so it is possible that they take part in the efferent side of this reflex; if this is the case no effect would be produced on the reflex by their division unless they contained its afferent fibres as well, since by the method employed the whole urethral resistance was examined and it is well known that by far the greater part of this is due to striped muscle supplied by the pudic nerves [3]. It has been shown previously [1] that the pelvic nerves exercise an inhibitory tone on the urethra and that the hypogastric nerves do not exercise a motor one, so if either of these nerves take any part in the reflex the pelvics would appear the more likely. Neither division of both pelvic nerves nor transection of the spinal cord, either in the lower thoracic or upper cervical regions, abolished this reflex, but they did modify it apparently in exactly the same way. After each of these operations the pressure the urethra resisted was found to be rather increased. As the pressure was raised contractions of the perineal muscles started if they were not present before, or became more numerous and stronger if they were. When the resistance was overcome the water escaped in a series of jets

with definite pauses between them which got longer as the pressure in the manometer fell, the result of which was that the fall of pressure took a longer time to be completed. The fall of pressure after the escape had started was usually less in degree than when the pelvic nerves and spinal cord were intact, at the same time it was quite easy to show that intermediate pressures were supported after these operations as before, therefore the reflex must still have been present. From these facts it must be concluded that both paths of this reflex are in the pudic nerves, with the possible reservation for the inhibitory fibres of the pelvics, already mentioned, taking a small part in it. The modification of the reflex in the same way by division of either the pelvic nerves or the spinal cord is possibly due to the fact that both these lesions abolish the reflex bladder contraction provoked by running water through the urethra. In describing the next reflex increased intravesical pressure will be shown to be associated with a reflex relaxation of the urethra. It is possible that the same may occur from contraction of the bladder even though it is empty. If this is the case the reflex contraction of the bladder which follows the running of water through the urethra would reinforce the direct reflex relaxation just described and division of either both pelvic nerves or the spinal cord would abolish this reinforcement. In a few experiments the effect on this reflex of injecting cocaine into the bladder was found to be of the same kind as that of dividing the pelvic nerves, but it was never of the same degree.

The fifth reflex was demonstrated by filling the urethral manometer to a pressure intermediate to that which was previously found to start an escape of water from the external meatus and that to which it fell when this escape occurred. The bladder was then filled by running in equal amounts every other minute as before. Directly the reactionary rise of intravesical pressure took place water started to run from the external meatus, and the pressure in the urethral manometer rapidly fell in consequence. The pressure to which the urethral manometer fell when a reflex was provoked by this means was usually lower than that to which it fell when water was simply run through the urethra with the bladder empty as described for the demonstration of the fourth reflex; this was presumably because the fourth reflex was actually occurring as well as the fifth and so reinforced the effect of the latter. The same effect was produced if both hypogastric nerves had been divided. Division of both pelvic nerves abolished the reflex. It was also abolished if 5 c.c. of 4 per cent. cocaine hydrochloride was injected into the bladder and left there twenty minutes or more; this effect was

not very easy to observe on account of the transitory nature of the effect of cocaine already mentioned. Division of the spinal cord in either the lower thoracic or upper cervical region did not abolish the reflex. After this operation the volume which previously caused a reflex contraction of the bladder had no effect on the urethra, and it was not till a greater increase in the distension of the bladder leading to a considerable elevation in the intravesical pressure had been produced that water escaped from the meatus and the pressure in the urethral manometer fell. When this had started, each subsequent increase of volume, leading of course to a still greater intravesical pressure, usually caused a further escape of water from the meatus and further fall of pressure in the urethral manometer. If the urethral pressure which just caused the escape of water from the meatus was determined by the method of gradual increments from zero already described when the bladder pressure was considerably raised by distension and then again after the bladder had been emptied, it was found that a greater pressure was necessary to force the urethra with the empty bladder than with the distended. From these facts seen after transecting the cord it is evident that the stimulus depends on the tension of the bladder wall and not on the degree to which it is stretched, and further that the reflex is continuously acting so long as this tension remains sufficient to excite it. Distension of the bladder produced contractions of the perineal muscles like raising the urethral pressure, though not to the same extent as the latter; the escape of water from the meatus occurred in jets after division of the cord and these could be seen to be synchronous with the intervals between these contractions. The afferent path of this reflex must therefore be in the pelvic nerves. The efferent path must be in the pudic nerves, since after division of these no effect could be produced on the residual urethral resistance by distension of the bladder.

From the foregoing remarks it is evident that the five reflexes demonstrated by the method under consideration are of such a nature that when one starts the others are brought into action automatically. If, as seems probable, though no evidence has been brought forward to prove it, the first reflex to occur is the contraction of the bladder provoked by distension, this contraction will of itself lead to relaxation of the urethra through the fifth reflex described; the urine running through the urethra will by itself lead to a relaxation through the fourth reflex described, and after a latent period of two or three seconds it will also lead to a contraction of the bladder as long as the stream lasts through the second reflex. The importance of the second

reflex for emptying the bladder is evident, as if it did not exist the lowering of intravesical pressure due to the escape of urine would abolish the stimulus provoking the first reflex and so the contraction of the bladder would not persist to the end. The use of the third reflex, which depends on the hypogastric nerves, does not appear very evident.

After transection of the spinal cord the first and second reflexes are abolished but not the third, fourth and fifth, hence the escape of urine from the urethra of such animals is due to reflex relaxation of the urethra through the fourth and fifth reflexes, the feeble and transitory contraction evoked through the third reflex being incompetent for the purpose. The constant presence of residual urine in these circumstances is no doubt due to the absence of the first and second reflexes. The conclusion of Goltz [6] that micturition can take place after spinal transection was based on the assumption that the reflex escape of urine found by Giannuzzi [5] and himself to occur some days after this operation was due to reflex contraction of the bladder, but he described no observations proving that this in fact occurred. To make certain that the stage of "spontaneous micturition," now so well known to occur in animals some days after a spinal transection, was not due to the reappearance of a reflex contraction of the bladder, the spinal cords of two female cats were divided respectively at the 8th and 9th thoracic roots under chloroform. After twenty-six and thirty-five days respectively, the cats were decerebrated and examined by the experimental method under consideration. In both of them the fourth and fifth reflexes were present. No sign of a reflex contraction of the bladder could be obtained by distending it. On raising the pressure in the urethral manometer, a moderate volume being present in the bladder, sharp transitory increases of bladder pressure up to 6 cm. took place both before and at the time water began to escape from the external meatus, but no sustained or greater rise of pressure was present. Hence, even when spontaneous micturition has become established, the two reflexes which lead to a strong and sustained bladder contraction are still absent.

The existence of these five reflexes affords a ready explanation for the fact that urine in normal cats, as shown by Griffiths [7], is held at the internal meatus, whereas in cats that have had a spinal transection, both pelvic nerves divided or the posterior sacral roots divided, it is held at the compressor urethræ [1]. The resistance of the urethra after division of the pudic nerves is greater than the pressure the bladder will tolerate without reacting: the greater part of this resistance, at any rate in females, must be due to the smooth

muscle of the posterior urethra, hence the posterior urethra will normally always be closed except when a reaction is occurring, i.e., during micturition. After any of these three lesions mentioned, however, the reaction of the bladder to distension is abolished, hence it allows its pressure to rise to a much greater extent without urine escaping; the posterior urethra can then be forced but the compressor being stronger will hold the urine for a longer time till the pressure has increased still further. The same effect should be produced by cocainizing the bladder, but as already stated this was not found to be the case; the explanation of this discrepancy would appear to be that during the time necessary to fill the bladder and take the X-ray the effect of the cocaine had, at any rate partly, worked off.

(4) Micturition in Decerebrate Cats.

The passage of urine in a forcible stream very commonly occurs five to ten minutes after decerebration; in such cases there is nearly always a distinct, though often small, quantity of urine left in the bladder. This residual urine, from what has been said of the effect of chloroform on reflex micturition, can be accounted for by the cats not having recovered from the chloroform at this time. This passage of urine is such a frequent occurrence after decerebration when there is any quantity of urine in the bladder at the time that it is impossible to doubt that it is due to the operation; from its frequency it appears to be due to the removal of an inhibitory influence arising in the part of the brain removed, and not to any secondary result of the operation such as bleeding into the posterior fossa.

Eight cats were decerebrated—all but one under aseptic conditions—and after arrest of bleeding the skull cavity was filled with paraffin wax by Bazett's method [2] and the wound closed. As it was necessary to tell when the cats passed urine, they could not be kept in a bath as was done by Bazett. They were, therefore, put in a closed cage in which a number of electric lights were fixed. The rectal temperature was taken periodically and the temperature of the cage altered when necessary by altering the number of lights burning. The experiments were all made in July or August. The duration of decerebrate life varied from 4 to 40 hours and averaged 16 to 17. The cage was placed on a tray which sloped towards its centre where it was perforated, a receiver placed under the perforations showed whether urine had been passed or not. In six of the eight cats micturition appeared to be performed normally. It

took place nineteen times and was actually seen in four of the nineteen. The urine was passed in a stream, finishing with a few jets associated with perineal contractions. Palpation of the abdomen after showed the bladder to be either empty or only to contain a small volume of urine such as might have accumulated in the interval of time since it was last looked at. The volume passed at each micturition varied from 10 to 120 c.c. and averaged 47 c.c., which is rather below the average quantity passed at a single micturition by a normal cat. The cat, which lived forty hours after decerebration, weighed 3,170 grm. and micturated seven times, the volume passed at each micturition varied from 34 to 70 c.c. and averaged 53 c.c. There, therefore, seems to be a slight degree of frequency of micturition in decerebrate cats, but the act of micturition itself is performed normally. In the other two cats micturition was not normal in that there always appeared to be residual urine about 5 to 10 c.c. in one and about 10 to 20 c.c. in the other. In both the amount of urine passed at each micturition was very small, 5 to 15 c.c. in one and 2 to 12 c.c. in the other. When micturition occurred it was passed in a stream in one, but it was never actually seen in the other.

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LESIONS OF THE POSTERIOR TIBIAL NERVE.

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AMONG the injuries to peripheral nerves resulting from gun-shot wounds, those most frequently overlooked, in the writer's experience, are lesions of the posterior tibial nerve. By the posterior tibial nerve is meant that part of the continuation of the internal popliteal nerve which lies between the lower border of the popliteus muscle above and the hollow behind the internal malleolus below. With injuries to the internal popliteal nerve itself (i.e., that portion of the internal division of the sciatic nerve lying above the lower border of the popliteus) this communication does not propose to deal; the resulting paresis of the calf muscles alone usually draws attention at once to the nature of the lesion. With the posterior tibial nerve, however, the case is different; the motor disability is relatively slight, more particularly when the lesion is below the origin of the nerve branches supplying the flexor longus hallucis and flexor longus digitorum muscles. The patient usually appears complaining of pain or numbness in the foot; it is seen that he is able to plantar-flex the foot at the ankle and also to flex the toes. The condition is often termed "functional" and the patient labelled "neurasthenia." Many of the sixty-four cases on which the following observations are based were received with such a diagnosis. When any penetrating wound of the calf or ankle has occurred, therefore, it is important to make a careful examination of the foot with regard to the presence or absence of sensory changes over the plantar area, and of paresis, atrophy or abnormalities of electrical reaction in the intrinsic muscles of the foot.

Lesions of the posterior tibial nerve, among nerve injuries generally, are somewhat uncommon. In 1,688 injuries to peripheral nerves, I met with sixty-four posterior tibial nerve lesions. This incidence is higher than that in both Purves Stewart's and Evans's series of 520 cases of nerve injury—four posterior tibial lesions (0·76 per cent.)—

and in Tinel's series of 639—eighteen posterior tibial lesions (2·8 per cent.).

To review briefly the anatomy of the posterior tibial nerve. From its origin at the lower border of the popliteus muscle, where the internal popliteal nerve ends, the posterior tibial nerve lies on the tibialis posticus muscle and the tibia, occupying with the posterior tibial vessels a sheath in the intermuscular septum which divides the superficial from the deep muscles of the back of the leg. During its course, the nerve supplies muscular branches to the flexor longus hallucis and flexor longus digitorum; the branches of supply to the two heads of gastrocnemius, soleus and tibialis posticus and the communicans tibialis are usually given off from the internal popliteal nerve in the lower part of the popliteal space. The soleus and tibialis posticus are often supplied by a common trunk, but occasionally the nerve to tibialis posticus may arise separately from the upper part of the posterior tibial nerve. As regards cutaneous branches, the internal calcaneal nerve is given off near the termination of the posterior tibial trunk; it pierces the internal annular ligament and is distributed to the skin of the heel and the posterior part of the sole. The posterior tibial nerve then terminates by dividing into internal and external plantar nerves beneath the internal annular ligament, the former nerve supplying flexor brevis digitorum, abductor and flexor brevis hallucis, the first lumbrical and the skin of the inner part of the sole as far as the mid-line of the fourth toe. The external plantar nerve supplies accessorius, the interossei, abductor and flexor brevis minimi digiti, the three outer lumbricals, adductor hallucis and the outer part of the skin of the sole from the mid-line of the fourth toe. The plantar collaterals supply, by means of dorsal branches, the dorsal surface of the ungual phalanges.

The internal plantar nerve is homologous with the median nerve of the hand and the external plantar with the ulnar.

I.—Syndrome of Complete Interruption.

The nerve fibres of the lower or peripheral segment of the nerve are entirely cut off from those of the upper or central end. This separation occurs for the reason that the nerve is completely severed, extremely compressed, or owing to the formation of a fibrous cicatrix as the result of tearing or bruising of the nerve.

Even when the nerve is wholly divided and the skin of the plantar area completely anæsthetic and analgesic, considerable pain in the sole

of the foot on walking may be the main symptom. The source of the pain is the end-bulb of the upper segment, the sensation being referred to its area of distribution, a point sometimes lost sight of. On the other hand, the complaint may be "numbness and weakness" in the foot, especially on exertion.

The physical signs of complete interruption are as follows:—

Sensory disturbances.—There is loss of sensibility to light touch (cotton wool) and pin-prick over the entire sole of the foot, the area of loss extending laterally to about half an inch beyond the margin of the

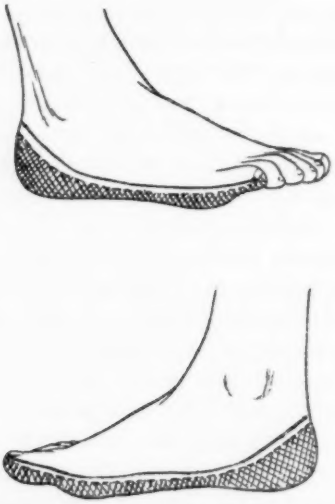

FIG. 1.—The area of loss of cutaneous sensibility with complete interruption of the posterior tibial nerve. The upper limits of loss of sensibility to cotton wool are represented by the continuous line; over the shaded areas sensibility to pin-prick is also lost. Loss of both cotton wool and pin-prick sensibility is continued over the entire sole of the foot. From a case (Case 1) in which at subsequent operation the nerve was found to be completely severed.

sole; the area of anæsthesia to light touch is, of course, slightly more extensive than that of analgesia to pin-prick. On the inner side of the foot the margin of both anæsthesia and analgesia is well defined; on the outer side adjoining the external saphenous area it is less definite. In some diagrams of the cutaneous area supplied by the posterior tibial nerve through its calcaneal, internal and external plantar branches, the lateral limits of the area are shown parallel to the margins of the foot throughout its length. I have invariably found, however, that in complete interruption of this nerve the cutaneous loss of sensation

involves a considerable portion of the lateral and posterior aspects of the heel (*vide* fig. 1). Owing to the relative thickness of the skin of the plantar area, sensation to cotton wool is often difficult to estimate, but this is not usually the case at the margins of the foot, to which particular attention should be directed. The dorsal surfaces of the outer four toes are usually described in posterior tibial nerve lesions as being insensitive to light touch, while no loss of sensibility to pin-prick is found over either their dorsal or plantar surfaces (fig. 2 A). While this distribution occasionally holds good, I have nevertheless found considerable variations. For instance, in one case in which subsequent operation showed the posterior nerve to be completely severed, loss of

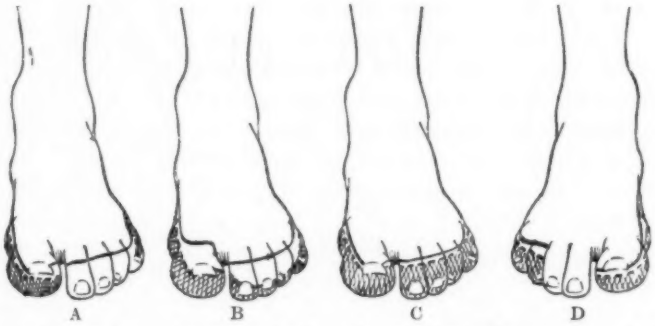

FIG. 2.—Illustrating variations in the loss of sensibility (to light touch and pin-prick) over the dorsal surface of the toes in complete interruption of the posterior tibial nerve. The continuous lines represent the upper limit of loss of sensibility to light touch (cotton wool); sensibility to pin-prick is also lost over the shaded portions. The loss to both forms of sensibility is continued round the anterior extremities of the toes to the plantar aspect in B and C and around the first, fourth and fifth in D. In A there is no loss of sensibility to pin-prick over the plantar surfaces of the outer four toes, but in D this form of sensation is lost on the plantar aspects of the third and fourth toes, but not of the second and third.

sensibility to pin-prick was present over the plantar surfaces of the toes and extended round their extremities to half way down the nails; loss of sensation to cotton wool was also present on the dorsum of the hallux, which is unusual (fig. 2 B). In a second case, the loss to pin-prick extended down the dorsal surfaces of the outer four toes as far as the proximal inter-phalangeal joints (fig. 2 C), and in a third case of complete division of the nerve, the outer two toes were insensitive to cotton wool and pin-prick stimuli both on their dorsal and plantar aspects, while the second and third toes exhibited normal sensibility on their dorsal surfaces, sensibility to cotton wool only being lost on the plantar (fig. 2 D). Also, in my experience, it is more usual to find

sensibility to pin-prick absent than present over the plantar aspect of the toes. In most cases I have found no loss of deep pressure sense; in two cases only was there apparent loss of deep sensibility over a small area of the plantar aspect of the heel.

Motor disturbances.—On inspection, it is found that there is usually hyper-extension of the proximal phalanges of the outer four toes and acute flexion of the second and third phalanges; this occurs owing to paralysis of the interossei which normally flex the toes at the metatarso-phalangeal joints and extend at the interphalangeal joints. The plantar muscles are paretic and wasted, the concavity of the plantar arch thus being exaggerated; in my experience, however, any marked degree of pes cavus is seldom met with. When the lesion lies below the origin of flexor longus hallucis and flexor longus digitorum, flexion of the toes is well performed owing to the action of these undamaged long flexors, but in many cases the degree of flexion is not so great as is possible in the toes of the normal limb; in others there is no difference. The degree of extension of the toes is often somewhat limited as a result of paralysis of the interossei and, for the same reason, they cannot be separated. The ankle-jerk is usually much diminished or is absent.

When the lesion lies above or involves the branches supplying flexor longus hallucis and flexor longus digitorum, in addition to the changes described above as resulting from complete interruption of the nerve at the lower level, voluntary flexion of the toes is impossible. The patient also has some difficulty in rising on the toes of the affected leg, but he is not entirely unable to do so.

Electrical changes.—In most works on medical electricity and nerve injuries, the motor points of the muscles supplied by the plantar branches of the posterior tibial nerve are not mentioned. They are therefore shown on fig. 3. Prior to testing the electrical reactions, the foot should be soaked in warm water for ten to fifteen minutes. In a complete lesion, on stimulating the posterior tibial nerve behind the internal malleolus with faradism, no response will be evoked in the plantar muscles. Similarly, when the active electrode is placed on the motor points of the various muscles supplied by the nerve, no contraction will occur. With galvanism, a response can usually be elicited in flexor brevis digitorum, abductor hallucis and in the interossei, the degree and nature of the contraction varying with the length of time that the nerve lesion has existed; in complete lesions of some months standing, the reaction is very sluggish and A.C.C. is greater than K.C.C.

With the condenser set—which I have invariably used for testing reactions in nerve injuries[1]—a response cannot usually be obtained at 100 volts in a normal flexor brevis digitorum and abductor hallucis with any stop under No. 6 (0·10 microfarads) and in normal interossei under No. 5 (0·083 microfarads) Lewis Jones scale. In complete lesions of the posterior tibial nerve, no response can usually be elicited in the affected muscles even with the highest stop—No. 12 (4·0 microfarads) at 100 volts.

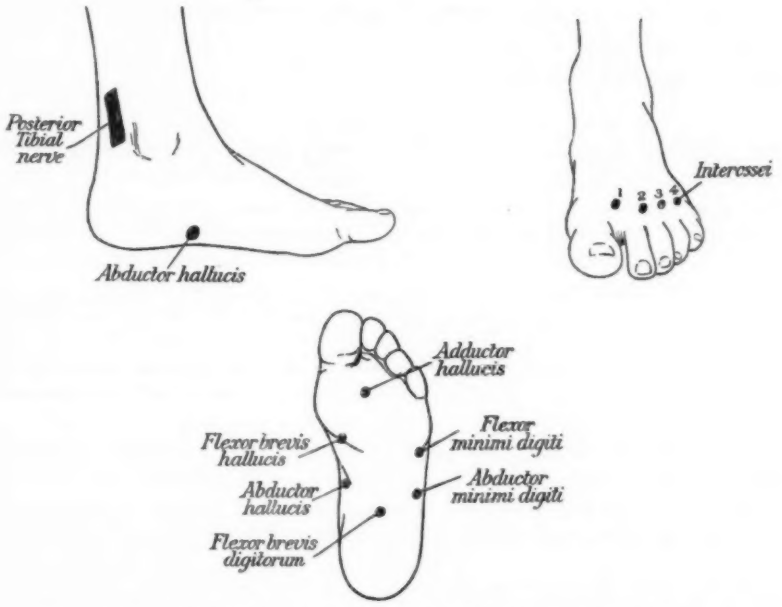

FIG. 3.—The motor points of the muscles supplied by the posterior tibial nerve.

In cases in which the flexor longus hallucis and flexor longus digitorum are involved, the response to faradism is absent in these muscles, and with the condenser a reaction will not, as a rule, be obtained with any stop below No. 10 (2·0 microfarads at 100 volts).

Trophic disturbances may be present in the form of scaly desquamation or mechanical ulcers over the sole of the foot; the latter lesions usually occur on the heel or at the base of the great or little toes.

[1] *Brit. Med. Journ.*, September 11, 1920.

Of the sixty-four cases of posterior tibial nerve lesions in my series, in sixteen interruption of the nerve was complete. Of these sixteen cases, in twelve the site of the injury was below the point of origin of the branches to flexor longus hallucis and flexor longus digitorum; in three it was above this level, and in the remaining case the injury involved the nerve at the level of the origin of the branch to flexor longus digitorum, the flexor longus hallucis, which is supplied from a slightly higher level, escaping and showing good voluntary movement and normal electrical reactions.

The following cases illustrate complete interruption of the posterior tibial nerve below the branches to the flexor longus hallucis and flexor longus digitorum muscles :—

Case 1.—C. McC., seen May, 1918, had received a bullet wound through the lower part of the leg in June, 1917; owing to sepsis, three months elapsed before the wound healed. No nerve injury was suspected. He was sent up for examination with the statement: "This man professes that he cannot carry on and no reason can be found." The complaint was one of numbness in the foot, a sensation as of pins and needles above the ankle and consequent difficulty in marching.

The scar of the entry wound was seen just behind the postero-internal border of the tibia, four inches above the internal malleolus, the exit scar being situated immediately external to the tendo Achillis, three inches above the external malleolus. There was cutaneous anæsthesia and analgesia over the entire plantar area. All voluntary movements at the ankle-joint were normal and the patient was able to flex the toes, the degree of flexion, however, being less than in the normal foot. Flexor longus digitorum and flexor longus hallucis responded normally to faradism, but no reaction was obtained in flexor brevis digitorum, abductor hallucis, nor in the interossei; these muscles also failed to respond even on No. 12 stop (4·0 microfarads) at 100 volts with the condenser set.

At operation, performed two days later, by Major Cecil Rowntree, the nerve was found densely adherent in firm fibrous tissue and, on dissection, was shown to be completely divided, an interval of half an inch separating the two extremities.

Case 2.—A. B., seen September, 1918, had been wounded by a piece of shrapnel in September, 1917, the missile entering three inches above the internal malleolus and leaving over the fibula, two inches above the external malleolus. The latter bone was fractured and owing to sepsis the wound had not healed until June, 1918, when he was discharged from the Army. No nerve injury had been diagnosed.

On examination, the patient complained of "burning pain," aggravated by walking, in the sole of the foot and above the back of the heel. There was

cutaneous anæsthesia and analgesia over the entire plantar area (fig. 2 B) and percussion over the entry wound gave rise to pain of a tingling character referred to the sole of the foot. All movements at the ankle-joint were normal; the toes could be well flexed but extension was somewhat limited. The plantar muscles were paralysed and atrophied. No response could be obtained with either faradism, galvanism or on the highest stop (No. 12—4·0 microfarads) of the condenser at 100 volts.

At the subsequent operation, the posterior tibial nerve was found involved in dense scar tissue, which, on being dissected away, revealed an upper bulbous extremity united to the lower segment of the divided nerve by a thin fibrous cord an inch long. After removing the upper end-bulb and incising the lower fragment until nerve fibres were seen, it was found impossible to bring the two severed ends of the nerve together. Consequently, the operator turned down a flap from the upper segment of the nerve, joining it to the lower—a useless procedure if regeneration is to occur. When the patient was examined eighteen months later, all the signs of a complete posterior tibial nerve interruption were still present, there being no evidence whatever of regeneration.

II.—Syndromes of Incomplete Interruption.

Incomplete interruption of the nerve functions may occur as the result of partial division, infiltration by interstitial traumatic hæmorrhage, or by involvement in and compression by cicatrical fibrous tissue or a fibrous band.

The sensory changes consist of loss of sensibility to cotton wool and pin-prick over the whole or only a portion of the plantar area, or, more frequently, mixed anæsthesia and over-reaction. The external plantar area is invariably the more anæsthetic, the internal plantar area often being the site of over-reaction. Varieties of the sensory changes that occur are illustrated by fig. 4.

In lesions of the inferior part of the nerve, there is more or less paresis and wasting of the plantar muscles and interossei, with diminished or absent reaction to faradism. With the condenser set, reactions are obtained on stops from No. 9 (1·0 microfarad) to No. 12 (4·0 microfarads) at 100 volts.

When the nerve has been damaged at the higher level, there is, in addition to the changes mentioned above, paralysis or weakness of the long flexors of the toes; in some cases voluntary movement in flexor longus hallucis is retained while flexor longus digitorum is paretic, and vice versa. In one of my cases, the lesion was complicated by a hysterical paralysis of the entire limb; the condition was easily differentiated by the extent of the paralysis and the faradic reactions.

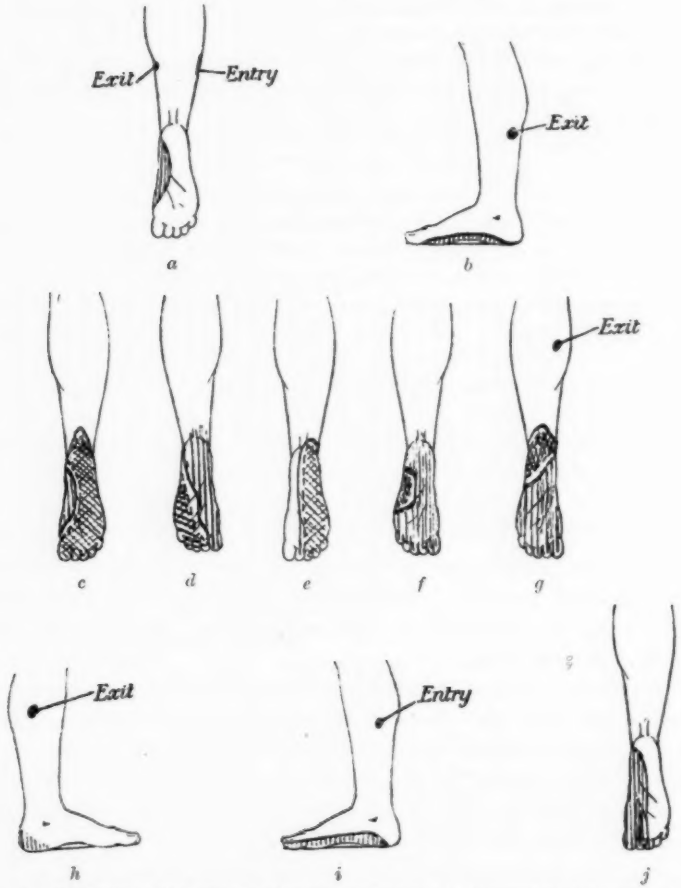

FIG. 4.—Varieties of the sensory changes encountered in incomplete interruption of the posterior tibial nerve. Over-reaction is represented by vertical lines. Shaded areas represent loss of sensibility to cotton wool and pin-prick, while the continuous lines just beyond shaded areas indicate the limit of loss of sensibility to cotton wool. *a* and *b* = over-reaction to pin-prick, with diminished sensation to cotton wool, over a portion of the internal plantar area (Case 5). *c* = Loss of sensibility to cotton wool and pin-prick over the greater part of the plantar area, a small area on the inner side (internal plantar) showing over-reaction. *d* = loss of sensibility to cotton wool and pin-prick over the external plantar area with over-reaction to pin-prick over the internal plantar and calcanean areas. *e* = Loss of sensibility to cotton wool and pin-prick over the external plantar and external portion of the calcanean area, the remainder of the area of posterior tibial nerve supply being normal. (At operation it was found that the outer side of the nerve was thickened and adherent to the adjacent tissues). *f* = A small area of loss of sensibility to light touch and pin-prick in the external plantar area, the remainder of the sole showing over-reaction. *g* = Loss of sensibility to cotton wool and pin-prick over the calcanean area, the external and internal plantar areas showing over-reaction. *h* = Over-reaction to pin-prick on the internal aspect of the calcanean areas. *i* and *j* = Over-reaction to pin-prick, with diminished sensation to cotton wool, over the internal plantar area, other areas being normal (Case 6).

The electrical reactions vary with the degree of the lesion; with faradism it will sometimes be possible to obtain a sluggish response in the long flexors but not in the plantar muscles or interossei. With the condenser, reactions are elicited on stops varying from No. 7 to 10 (0·25 to 2·0 microfarads) at 100 volts in the long flexors, and No. 9 to 12 (1·0 to 4·0 microfarads) in the intrinsic muscles of the foot at 100 volts.

In practically all cases of incomplete lesions of the posterior tibial nerve, there are symptoms of more or less irritation of the nerve trunk. The irritation gives rise to painful sensory disturbances varying from simple neuralgia to intense causalgia. In my experience, however, the posterior tibial nerve is less often the seat of true causalgia than the internal popliteal nerve. Spontaneous pains, described as shooting, burning or pricking in character, may be complained of. The pain is intensified by movement, muscular contraction, extremes of heat and cold, and by mechanical stimulation of the plantar area. In some cases there is cutaneous anæsthesia over the sole of the foot, but deep pressure gives rise to considerable pain; in others, diminished sensibility to light touch (cotton wool) coexists with over-reaction to pin-prick. Friction, firm tactile stimuli or pin-pricks applied to a plantar area of over-reaction results in a diffuse and radiating painful sensation of a burning or rending character, the stimulus being only imperfectly differentiated and localized by the patient. When, in an incomplete lesion of the posterior tibial nerve, there exists both over-reaction and anæsthesia over the skin of the plantar area, it is almost invariably the internal plantar that exhibits the over-reaction. This is illustrated by the following case, which is also of interest in showing over-reaction of the lateral part of the heel, which has previously been referred to as being within the area of posterior tibial supply.

Case 3.—A. M. C., seen April, 1918, was wounded through the ankle by a bullet in April, 1917, exactly a year previously. Since being wounded he had complained of pain in the instep and across the base of the toes, becoming more severe on attempting to walk.

On examination, the entry scar was seen an inch above and behind the tip of the internal malleolus, the exit being just above the external malleolus. The greater part of the plantar area was insensitive to both cotton wool and pin-prick, but on the inner side of the sole and heel there was an area showing marked over-reaction to pin prick but not to cotton wool (fig. 5). Flexion of the toes was well performed. The plantar muscles and interossei all failed to respond to faradism, and only reacted on No. 12 stop (4·0 microfarads) at 100 volts with the condenser set. A diagnosis of adhesion in fibrous tissue,

with incomplete interruption and slight irritation, was made and exploration advised.

At the operation performed a few days later by Major R. Jocelyn Swan, the posterior tibial nerve was found tightly compressed and bound down by a band of fibrous tissue in the track of the missile; it was also adherent to the surrounding tissues and exhibited slight fusiform enlargement. Prior to freeing the nerve, faradic stimulation of its trunk above the lesion gave rise to a moderate response in the plantar muscles.

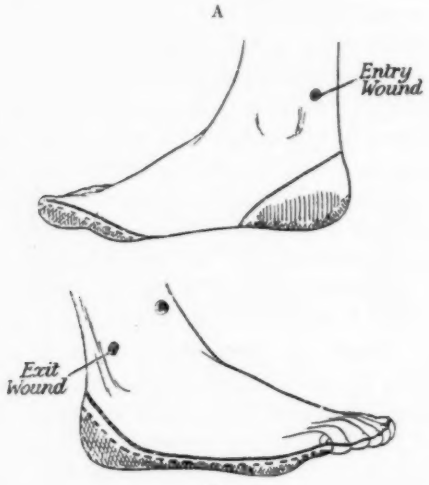

FIG. 5.—Changes in cutaneous sensibility in a case of incomplete interruption with irritation of the posterior tibial nerve (Case 3). The continuous lines in A and B represent the limits of loss of sensibility to cotton wool. The shaded portions are, in addition, insensitive to pin-prick. Over-reaction to pin-prick is represented by vertical lines.

In other cases, the symptoms and signs of irritation predominate, and the whole or greater part of the plantar area may exhibit intense over-reaction. Although voluntary movements are often retained in such cases, there is almost always some change in the electrical excitability of the muscles. The skin of the sole may appear smooth, glossy and pinkish in colour; other trophic disturbances may be present in the form of profuse sweats or scaly desquamation. The actual lesion is often found to consist of slight adhesions between the nerve and the surrounding tissues, the freeing of which affords considerable relief, or the nerve exhibits fusiform enlargement—due to interstitial hæmorrhage—at the site of injury. The following example illustrates a case

in which the nerve was the seat of painful neuralgia, the whole plantar area showing over-reaction the freeing of adhesions greatly relieved the pain.

Case 4.—R. A., seen June, 1917, had been wounded through the calf by a bullet four weeks previously; for the past three weeks he has complained of intense pain in the sole of the foot, aggravated by any movement or upon the limb being touched, and which keeps him awake night after night; drugs have given little or no relief.

On examination, the small entry and exit wounds were seen respectively on each side of the junction of the middle and lower third of the shank. The whole of the plantar area exhibited marked over-reaction to painful stimuli. He flexed the toes somewhat weakly and reluctantly, stating that the movement gave rise to considerable pain. The long flexors of the toes responded normally to electrical stimulation, but he was unable to bear any attempt at testing the plantar and interosseous muscles.

The nerve was exposed ten days later by Major R. Jocelyn Swan and, in the track of the bullet, the nerve was found adherent to the tibialis posticus muscle posteriorly; there was also slight fusiform swelling of the nerve. It was freed from the adhesion and wrapped in cargile membrane.

When seen a month later, the patient still complained of slight pain in the sole of the foot (no doubt from the interstitial hæmorrhage), but stated that "it was nothing compared with what it was before"; the pain did not interfere with his sleep, and he was able to get about well.

Occasionally, a portion only of the plantar area exhibits over-reaction, the remaining being normal. In such cases it is almost invariably the internal plantar area that is affected, as in the following case:—

Case 5.—P. K., seen December, 1918, was wounded through the lower half of the calf by a shrapnel bullet in June, 1918. Ever since he has complained of pain in the sole of the foot; the pain experienced on putting the foot to the ground causes him to limp.

On examination, there was no actual sensory loss, but a portion of the internal plantar area showed diminished sensibility to cotton wool but marked over-reaction to pin-prick (fig. 4a). All movements at the ankle were normal and he was able to flex the toes, though performing the movement slowly and carefully. Flexor longus hallucis and longus digitorum reacted normally to faradism; the plantar muscles responded only sluggishly to faradism, and on no stop below No. 10 (2·0 microfarads) at 100 volts with the condenser.

It is probable that in this case the nerve was adherent on its inner side to the adjacent tissue (as in Case 6) and would have been greatly relieved by operation; unfortunately the patient was lost sight of.

The following case is of interest as it is one of the few that I have

seen in which causalgic symptoms were due to a localized adhesion of the nerve to its sheath and in which all pain was completely relieved by the freeing of the adhesion:—

Case 6.—P. W. was seen two months after having received a penetrating bullet wound through the calf. At the time of being hit, he had experienced acute pain in the foot, and for three days had complained of pain extending from the level of the wound down the back of the leg to the toes; following this the pain persisted in the sole of the foot only and would become much worse towards the end of the day. It was aggravated by any movement, cold and extreme heat, and the patient was reluctant to allow the foot to be touched.

On examination, there was marked over-reaction to pin-prick over the greater part of the internal plantar area and extending on to the inner margin of the foot; this area was somewhat diminished in sensibility to light touch (cotton wool). The external plantar and calcanean areas were normal (fig. 4, i and j). All movements at the ankle-joints were good but the patient was unable to flex the toes. Faradic excitability was diminished in the long flexors and scarcely present in flexor brevis digitorum. The former muscles reacted on No. 8 stop (0·5 microfarad) at 100 volts, but not below, with the condenser, while flexor brevis did not react below No. 9 (1·0 microfarad).

At operation, performed a few days later by Major R. Jocelyn Swan, the track of the bullet was traced towards the nerve, when it was found that about 1 inch above the level of the entry wound the nerve sheath was much thickened; on opening the sheath the nerve was found adherent on its inner side only; this adhesion was broken down. For about ¾ inch at this level the nerve appeared slightly more purplish and translucent than above and below. It was wrapped in cargile membrane and the wound closed.

The patient was seen about three weeks later and stated that all pain disappeared about fourteen days after the operation. There was no cutaneous over-reaction present and the toes showed good voluntary flexion. When re-examined a month later, beyond the presence of the scars, no abnormality could be detected.

Unfortunately, it cannot be claimed that all cases of causalgia yield so promptly to the liberation of the nerve from adhesions; not infrequently, no such adhesions are present, the nerve merely showing slight fusiform enlargement or a bluish and translucent zone at the site of injury. The intraneural injection of 90 per cent. alcohol or even section of the nerve above the level of injury may be necessary.

Of the sixty-four cases of posterior tibial nerve lesions, incomplete interruption had occurred in forty-eight. Of these, in thirty, flexor longus hallucis and flexor longus digitorum were involved, the injury in the remaining eighteen being below the origin of their branches of supply.

SUMMARY.

Injuries to the posterior tibial nerve are frequently overlooked, no doubt because they cause no striking paralysis, all movements at the ankle-joint being normal. More especially do they escape recognition when the lesion lies below the point of origin of the branches supplying flexor longus hallucis and flexor longus digitorum muscles, as in such instances power of flexing the toes is retained. In all wounds involving the calf where there is a complaint of pain or weakness in the foot, a systematic examination must be made of sensation in the plantar area and of the functions of the intrinsic muscles of the foot.

When the posterior tibial nerve is damaged, changes in cutaneous sensibility—varying from over-reaction to complete cutaneous anæsthesia according to the degree of injury—will be found over the sole, at the margins of the foot, and over the posterior and lateral aspects of the heel. There is also paresis and often atrophy of the plantar muscles and interossei, the toes being hyperextended at the metatarso-phalangeal joints and flexed at the interphalangeal joints. Flexion of the toes, and occasionally extension, may be less complete than in the opposite and normal limb.

When the site of injury is above or involves the branches of the posterior tibial nerve supplying the flexor longus hallucis and flexor longus digitorum muscles, flexion of the toes is either much diminished or is entirely absent. The ankle-jerk is also reduced or impossible to elicit. On faradic stimulation of the posterior tibial nerve on the inner side of the tendo Achillis, the response in the plantar muscles will be greatly diminished or absent. Similarly, stimulation of the motor points of the affected muscles shows reduced or absent reaction to faradism, a response only to the higher condenser stops—No. 8 (0·5 microfarad), to No. 12 (4·0 microfarads) at 100 volts, Lewis Jones scale—or, in complete lesions, no reaction even with the highest capacity, No. 12 (4·0 microfarads). The galvanic reactions vary with the degree of the interruption of the nerve functions.

When sensory disturbances of long standing are the predominant feature, exploration of the nerve should be considered. If the nerve shows complete interruption, the end-bulb of the upper segment should be removed or the fibrous cicatrix excised and end-to-end suture performed. In incomplete lesions exhibiting neuralgic symptoms the freeing of an adherent nerve will usually result in considerable relief.

Proceedings of the Section of Neurology of the Royal Society of Medicine.

Held December 9, 1920.

President—Dr. Ernest S. Reynolds.

Tabes: Its Early Recognition and Treatment.

By E. Farquhar Buzzard, M.D., F.R.C.P.

Dr. Buzzard first narrated the history of two patients suffering from tabes dorsalis. He then continued as follows:—

Tabetic Pains.

In order to justify a diagnosis of tabes in the absence of physical signs it is necessary to state one or two facts about which there can be little disagreement. In the first place, the disease is one which attacks the afferent paths of the nervous system, and it is only reasonable to suppose that its earlier manifestations are of a subjective character—in other words, that complaints indicating a disorder of function precede those which are clearly due to structural defects. In the second place, it will be agreed that in a large proportion of cases of unmistakable tabes careful inquiry will elicit a history, often going back five, ten, or more years, of pains which have been regarded as neuritic or rheumatic in origin, and have not been associated with any other disability. Not once but a score of times have I asked a tabetic patient whether he has suffered from pains. After repeated denials I have altered the wording of my question and inquired for rheumatism. "Oh, yes, I have suffered from rheumatism for years." "What do you mean by rheumatism?" "Why, sharp, shooting pains in my limbs." This conversation shows how little the patient has associated his pain with the disability for which he is seeking advice, and incidentally how few patients understand the language they speak.

Granted that pains can be, and often are, the earliest signs of tabes, can they be distinguished from pains of other origin? I would answer this question in the affirmative and even go so far as to say that the pains of tabes described by a patient who is able to translate his feelings into intelligible language are quite pathognomonic. The fact that they are called lightning pains and that patients often say that they shoot up and down their legs has been responsible for a very general misunderstanding of their true character. When the patient states that they shoot up and down the limb he does not mean that each pain shoots from the hip to the foot and vice versa. He means that during an attack he may have pains first in the thigh and a few minutes later in the foot. If he is asked to describe them in greater detail he will inform you by gesture and by word that these pains have an axis, so to speak, at right angles to the surface of the limb. They stab like a knife or a darning-needle going in, or they resemble the effect produced by taking up the flesh, pulling at it, and letting it go again. Further, the pains come not singly, but if not in battalions at any rate in platoons. They are rapidly repeated, several occurring in the course of a second or two, followed by a lull of longer or shorter duration. Bouts of paroxysms may last for a few hours to a few days and then cease for a time. Favourite sites for these pains are the heel, the inner aspect of the shin, and the inner aspect of the knee. The area within which they repeat themselves is small, described by the patient as the size of a crown-piece, or no bigger than the palm of the hand. Between the frequent paroxysms these areas are often tender to light touch, so that the patient does not even like the contact of his clothes. Many patients are quite certain that pains may be provoked by a cold draught, by a change of weather, or even by a glass or two of beer. Others are equally sure that the responsibility lies with fatigue or anxiety. In the early stages of the disease pain rarely occurs during active exercise, but later on walking may be brought to a full stop by a sudden and overpowering paroxysm.

Before leaving the subject of pains let me make it quite clear that those I have described are not the only ones suffered by tabetic patients. There are deep, gnawing, aching pains in the bones, and others which have not the same diagnostic value. Let me also emphasize the fact that tabetic lightning pains must be recognized by their characters and not by their severity. Many patients confess to what they call "niggling pains," trivial in respect to suffering, but identical in nature with the lightning type.

It would be surprising if there were not other subjective phenomena in early tabes, and attention may be drawn to the not uncommon complaint that hot or cold water has become disagreeable or unbearable, that the patient is obliged to have his bath at a temperature which is described as tepid. Not that the hot or cold water provokes pains, but that it gives rise to a sense of great discomfort. This often applies to the whole surface of the body, although pains may be absent or confined to the lower limbs. Similarly it is not uncommon to hear that the fingers are numb when it is quite impossible to determine any disorder of sensibility by tests. This, of course, is not pathognomonic of tabes.

Treatment.

The necessity for advising antisyphilitic treatment of tabetic patients is not now so urgent as it was a few years ago, but it may be doubted whether the principles of this treatment are yet sufficiently recognized. We are all asked: "How long must I go on with treatment before I am cured?" For many years my answer has invariably been, "For the rest of your life." I am never consulted about a primary chancre, but if I were my advice would be the same. It cannot be denied that we are not and never have been in a position to tell a patient that he has been cured of syphilis, and, as neurologists, we are now constantly seeing tabetic patients who have been cured of syphilis by the arsenical compounds, just as fifteen years ago we saw those who had been cured by mercury. I maintain that the only honest advice we, as medical men, can give to patients suffering from any syphilitic disease is to the effect that they should continue to have periodic courses of treatment for the rest of their lives. Tell them that prevention is better than cure, and that they must regard this precaution as a method of insurance. In my experience so many victims of tabes and other syphilitic nervous diseases have allowed valuable time to slip by owing to negative Wassermann reactions that my confidence in this test as a method of regulating treatment is shattered.

Conclusion.

Is there any lesson to be learned from the story of the two patients which I have presented to you? The first man was infected with syphilis fifteen years ago, and has undoubtedly suffered from tabes for twelve years, during eight of which he has been under treatment. To-day he would be passed as a first-class life by any medical officer of an insurance office who did not know his history. It would be unwise

and unjustifiable to say that he is cured, but it is difficult to believe that the natural progress of his disease has not been arrested. And it must be remembered that he wasted four years before his symptoms were recognized and treated. The second patient was more fortunate. Only six months were wasted, and the result of treatment was rapid and lasting, judged by the fact that he has now enjoyed six years of freedom from any kind of symptom. These are not the only cases of the kind which have come under my observation, but I regret to say that it is a rare experience to be asked to see a patient suffering from tabes whose symptoms have not been of long-standing.

It is obvious that the value of these observations depends on the reliance which can be placed on the description of pains given by patients, and on the criterion the description affords for purposes of diagnosis. In many cases the description is vivid and easily recognized. In others it can only be elicited by patient cross-examination, avoiding the employment of leading questions. After many years of critical study of this problem I am convinced that the lightning pains of tabes are, for practical purposes, pathognomonic, and that they can and should be recognized before they are many months old. Treatment initiated before physical signs are apparent is not only possible in many cases, but is likely to be attended by good results if persevered with to the end of the chapter.

The Effects produced by obscuring the Vision of Pigeons previously deprived of the Otic Labyrinth.

By Sydney Scott, F.R.C.S.

Two pigeons were shown, in whom successful operations had been performed, and Mr. Scott made the following remarks:—

The research was originally undertaken to ascertain whether pigeons whose labyrinths had been destroyed would in the course of time regain power of flight, which is temporarily lost by interfering with the labyrinthine functions. With permission of the Home Office the experiments were commenced in the autumn of 1919. Under general anæsthesia absolute alcohol was injected into the labyrinth with a hypodermic needle, which was passed through the tympanic membrane and made to penetrate the base of the columella into the vestibule. This procedure was carried out with the aid of a small

aural speculum and bright head light, as employed in the operation of incising the tympanic membrane in cases of otitis media in human beings, smaller instruments, of course, being required. In some pigeons only one labyrinth was destroyed, in others both were destroyed, either immediately or after an interval of some days. In the course of nine months or thereabouts, thirteen pigeons were operated upon. Several difficulties were encountered in the first series before it was found possible to keep the birds alive for sufficiently long periods to enable me to make observations on their powers of flight. One died from the anæsthetic alone. Another collapsed under chloroform, but recovered after inversion and artificial respiration. In another bird the base of the columella could not be felt and the operation failed on three separate occasions to produce any sign of labyrinth destruction, such as forced movements, with inco-ordination, and disturbance of gait, flight, or assumption of abnormal posture. Another bird died instantly at the moment of injection; too deep penetration and excessive quantity of alcohol injected would account for this sudden effect on the brain. Among two survivors, it was evident that the alcohol had acted on one labyrinth only in each case, though both ears had been injected. In these two birds, on recovery from the anæsthetic, the forced movements and altered behaviour was of such a character that it was evident that the right labyrinth was rendered functionless in one bird, and the left labyrinth in the other. Of those which survived the actual operation, four died between the seventh and the eleventh days, apparently of faulty nutrition, though hand-fed after the operation. On this account it was decided to keep the birds for two months before operation to accustom them to be hand-fed daily during that interval, and afterwards as long as was found necessary. Eventually two successes were obtained, both birds surviving over nine months, during which time numerous observations were made on their behaviour and powers of flight, one with only one labyrinth destroyed, and in the other with both labyrinths successfully destroyed.

In order to preserve permanent records of the movements and behaviour of these birds about 1,400 feet of cinematograph film pictures were obtained at various intervals. These films were demonstrated.

BEHAVIOUR OF PIGEONS BOTH LABYRINTHS OF WHICH WERE DESTROYED.

July 17, 1920.—10.8 a.m.: Chloroformed. 10.10 a.m.: Absolute alcohol injected into left labyrinth. 10.12 a.m.: Absolute alcohol injected into the right labyrinth. 10.20 a.m.: Recovering from anæs-

thetic; tendency to hold head back, and when standing to fall directly backwards; irregular head nystagmus; no ocular nystagmus observable.

July 21, 1920.—Quite active. Tries to peck grain-food, but tends to pitch forwards when beak touches floor level. Appears to be trying to avoid pecking too far forwards. Misses grain by about a centimetre, eight to ten times, before picking up. Stands with wide base, but not asymmetrically. Within two months this bird walked about and could fly up to perch 2 ft. high. Marked tendency to overbalance backwards, always holding beak high in the air. When removed from cage flew straight in a room, but misjudged when landing, and pitched forwards on to beak. Wing movements symmetrical; the bird did not fly round in a circle, or appear eager to fly at all when released occasionally.

Effect of covering one or both Eyes of the same bird with a Hood.

When both eyes are covered with a hood, the bird remains motionless in whatever position it is placed. If standing, when eyes are covered the beak goes forwards, rests on the floor, or the bird overbalances backwards, turning a somersault till the head rests with neck flexed beneath its back on the floor. If the eyes be covered and the bird be laid out like a dead bird, it will remain motionless except for respiratory movement an indefinite time. It was found to behave in the same manner when in a dark room, the observer judging the bird's posture by feeling with the hand. When in the light—only one eye was covered—the bird walked around slowly in a circle, the centre of which was towards the open eye.

About six months after the injection this bird could fly from the floor up to a window, and though it never landed easily like a normal bird, it learnt how to prevent itself pitching forwards violently on to its head. It never learnt to peck accurately at grain, and though it attempted to feed itself early, and hand feeding became unnecessary in a few weeks, still it was found advisable to supply it with a cup of grain instead of with grain scattered on the floor. It evidently experienced difficulty in selecting its food.

Incidentally the bird appeared to be deaf, that is to say, it never showed the movements associated with startling noises, such as explosion of old electric light bulbs, as its normal or unilaterally defunct companions did. Moreover the movements of the head were more rapid and constant as if the bird were dependent solely upon vision, which it probably was. When hopping from ground to perch the

balancing on the perch was at first accompanied by swaying movements in an antero-posterior plane, the bird sometimes falling backwards (never forwards) off the perch.

Unilateral Destruction of the Labyrinth.

In marked contrast with normal birds and the birds with both labyrinths destroyed, the behaviour of those with only one labyrinth destroyed is markedly different and quite characteristic.

The following is an example: A blue chequer pigeon was injected in July, 1920, and survives in May, 1921.

July 17, 1920.—11.16 a.m.: Chloroformed. 11.20: Absolute alcohol injected into left labyrinth. 11.30: Recovering from anæsthetic and runs or flutters circus-wise to the left.

July 28, 1920.—Stands erect until disturbed; when attempts are made to handle the bird in its cage, it tries to evade the hand and flutters round to the left and drops the left side of the head to such an extent that the head is axially rotated until the lower beak becomes uppermost, and the bird holds its head with the vertex resting on the floor of the cage. To a less extent it is obvious that the left side of the body is also lower than the right. The bird remains still in this position, looking with its right eye at an observer who is standing on the bird's left side. In a minute or so the bird suddenly resumes its normal posture and stands erect. When removed from the cage the bird may walk straight a little way, but if pursued it spreads its wings and flutters to the left, eventually reaching some corner which arrests progress and there re-assumes the position of axial rotation of the head as described above. If the bird be held in the hand off the ground and coaxed to fly, it invariably goes in a circle, alighting on the ground, and shows no desire to resume flight. The bird never flies or hops up to its perch like the bird with both labyrinths destroyed. It sleeps on the floor of the cage.

One bird whose labyrinth had been destroyed on one side attempted to fly when released on the roof of a building, but on reaching the parapet its flight movements were definitely circus-wise, and it lost height, resembling the "spinning nose dive" of an aeroplane, landing in a heap on the ground 72 feet below. The bird appeared to be dazed but was not apparently injured, and lived for some few weeks afterwards. However, it was decided not to allow others to escape and possibly injure themselves in this way.

The Effect of obscuring the Vision in Pigeons devoid of one Labyrinth.

When a hood is placed on the head and both eyes are covered, the bird keeps still, with the head axially rotated to an extreme degree as previously described. When only the left eyeball is uncovered (assuming the left labyrinth is destroyed) the bird tends to hold the head in the normal erect position and walks slowly in a circle towards the left. When only the right eye is uncovered (the left labyrinth having been destroyed) the bird reassumes the axial rotation of the head with the left side downwards and the underbeak uppermost, while it excitedly executes rotatory movements to the left. Normal pigeons when hooded will walk about to right or to left or forwards, or stand on a perch, or sit at roost, but will not remain passive or overbalance themselves like hooded birds which have been deprived of one or both labyrinths.

CLINICAL MEETING HELD AT THE NATIONAL HOSPITAL FOR THE PARALYSED AND EPILEPTIC, QUEEN SQUARE, FEBRUARY 10, 1921.

President—Dr. Ernest Reynolds.

(1) *A Case for Diagnosis; possibly Tabes Dorsalis.*

By Dr. Tooth.

M. D., female, aged 35.

Family history *nil ad rem.*

Ten years ago transient attacks of loss of vision, which recurred several times during the next four years; none since then.

Five or six years ago began to have trouble in walking, tendency to walk quickly and fall forwards; in hospital in 1917 for ten weeks; improved for about twelve months, then trouble began to recur and has gradually got worse since.

Twelve months, some hesitancy in micturition.

Six months, girdle pain on the right side.

Twelve months ago legs gave way and she had complete right hemianæsthesia and hemianalgesia for a fortnight.

At Christmas, 1920, had anæsthesia and analgesia left lower limb, lasting six weeks.

Present state.—Optic discs physiologically pale; pupils react to light sluggishly; nystagmus in horizontal direction on extreme lateral movement,

especially to the right and in vertical direction on extreme upward movement. Sensibility to pin-pricks slightly diminished over right half of body and slightly less keen than normal all over. Deep muscle-pain sense in legs absent. Touch: localization of touch, passive movement, stereognosis good. Power slightly diminished in legs, and legs hypotonic. Co-ordination fairly good, slight tremor in hands on finger-nose test. Arm jerks, knee and ankle jerks absent; right abdominals present, left absent; right plantar slight flexor, left extensor. Gait unsteady. Rombergism present. Wassermann in blood and cerebrospinal fluid negative.

(2) *A Case of Acute Myelitis at about Seventh Dorsal Segment, with Optic Neuritis.*

For Dr. TAYLOR, by Dr. SHEPHERD.

G. S., female, aged 23. Woke up on July 15, 1920, with pain over the left eye; the sight went in that eye by the next morning and she had pains in the legs and back during the night. She was feverish and had headache the next day and the right eye became similarly affected, so that by the next day she was absolutely blind and was completely paralysed in both legs with analgesia and anæsthesia up to the waist, and retention of urine and fæces.

Legs were quite spastic for ten days and then the spasticity gradually went off and the legs became quite flaccid. Eyesight gradually improved, so that she can see quite well now. From October, 1920, incontinence of urine.

Physical examination.—Optic discs pale, especially the left, showing evidence of old optic neuritis. Complete paralysis of legs below waist (very slight flicker in toes only). Complete loss of sensation to cotton wool, pin-prick, temperature, from below the waist; the loss to pin-prick extends an inch higher than the loss to cotton wool, and in the area between these two upper levels hot and cold is wrongly appreciated. Arm jerks normal, knee and ankle jerks, abdominals and plantars absent.

Cerebrospinal fluid: albumin 0·6 per cent., N.A. strongly positive, no cells seen, Wassermann negative. Wassermann in blood negative.

(3) *Case of Intracranial Pressure (?) Tumour; Recovery without Operation.*

For Dr. TAYLOR, by Dr. SHEPHERD.

E. F., female, aged 28. At the age of 16 suffered with headache and loss of power in the hands and legs, especially the right side. Headaches became very severe and continuous and were accompanied by vomiting. She was admitted to the hospital in October, 1904. She had then marked papillœdema, but the headache and vomiting subsided soon after admission and the power gradually returned to the limbs.

About three months after admission she was able to be up and walking, but the arms and legs got weak again: she was very unsteady and had to go back to bed, so that she was still very weak about August, 1905. She gradually improved again and was able to be about in six months.

From that time until eighteen months ago she had been fairly well, with the exception of some tremulousness and weakness in the legs, especially in the right.

During the last eighteen months the right leg has become weaker.

No visual or sphincter troubles; only a very occasional headache; some tingling in the hands and feet during the last few months.

Optic discs normal; (?) slight nystagmoid jerking on looking to the right; otherwise cranial nerves normal. Movements of right leg much diminished with slight talipes equinus on right side. Abdominal reflexes active and equal, knee jerks and ankle jerks active and equal; clonus, right plantar inactive, left flexor. Walks dragging right leg.

(4) *Case of Friedreich's Ataxia.*

For Dr. TAYLOR, by Dr. SHEPHERD.

F. L., male, aged 19.

No family history. Patient has one sister and one half-brother.

Patient began to be unsteady in walking at age of 7 years; says he always fell upon his nose; this gradually became worse, legs weak and muscles gradually wasted, so that by age of 14 he was confined to a chair.

No change during the next three years, but has been feeling stronger during last two years. Deformity of the spine, said to have developed during last three years. Pes cavus present since childhood. No alteration noticed in speech.

Patient is rather facile boy of 19, not keen mentally, bodily nutrition bad, arms wasted. Nystagmus present, especially to right. Also slight shaking of head; speech slow and of scanning variety; motor power diminished in lower extremities and in trunk, with wasting in legs; no spasticity. Slight pes cavus, slight intention tremor. Deep reflexes absent; plantars extensor. Slight kyphoscoliosis. Wassermann negative in blood and cerebrospinal fluid.

(5) *A Case of Lethargic Encephalitis.*

By Dr. JAMES COLLIER.

Mary B., aged 20, was seized with intermittent frontal headache on December 26, 1920. On the following day her eyes became crossed, and she saw double and became very sleepy and ceased to talk or to move spontaneously. She had considerable trouble with retention of urine. She was not laid up, and attended her doctor regularly. She has remained in this condition since, and during this time her memory has been good; she has obeyed orders promptly and replied to all questions promptly and correctly. Her facial and bodily expression is one of complete statuesque immobility, in lying, sitting, or standing. She will remain in any awkward attitude in which she is placed for long periods. She never moves spontaneously except in response to the more urgent calls of nature. She does not turn over in bed. She presents no emotional expression, positive or negative. She never speaks

spontaneously. She obeys every word of command at once and correctly. She answers every question at once and correctly, and gives a correct and detailed account of her illness if she is catechized. In response to the usual salutation she answers always: "I feel quite well, thank you."

There is severe bilateral ptosis which is diminishing. Complete internal ophthalmoplegia with large pupils. The axes diverge in vertical parallelism. Downward movement absent. Upward movement almost absent with quivering. Lateral movements of about two millimetres with quivering. Vision very poor, but no change in media or fundus.

No other brain-stem paralysis.

On admission to St. George's Hospital, January 12, 1921, the left abdominal reflex was diminished and the left plantar was extensor. Now the abdominal reflexes are equal and the plantars are flexor. Some fibrillation was noticed in the right anterior tibial and peroneal muscles.

Under observation she has developed dropped toes and dropped in-turned foot on the right side with severe weakness of the external sciatic group of muscles, with increased tendon jerks.

The points of interest are the peculiar clear but immobile mental state and the condition of the right foot.

(6) *Case of Amyotonia Congenita.*

Shown by Dr. COLLIER.

V. C., female, aged 4½.

Child born normally. Difficulty in sitting up; has never walked, and only began to crawl ten months ago. Pain in foot on being stood up. Otherwise no complaint; no fits nor injuries. Bright child, teeth and talking at normal age. Ten months ago had headache, vomiting and screaming for three days; otherwise no illnesses. Clean in habits. Father died of consumption. Three siblings dead—one of meningitis, at 5 years; two at 4 and 3 months, having been sickly from birth: one alive, aged 13, and healthy.

Child of poor development, good intelligence, and unusually good-tempered and uncomplaining. Stands with support, hips and knees being flexed from contractures of muscles, and feet slightly plantar-flexed. Muscles of lower limbs only of poor tone and somewhat wasted, but moderate power. Bears stretching of contracted muscles well, and has improved considerably since admission. Knee and ankle-jerks present. Abdominal reflexes present. Plantars flexor.

(7) *Case of Graves' Disease; (?) Paralysis Agitans.*

Shown by Dr. COLLIER.

M. B., female, aged 26.

Lump on throat noticed eight years ago; has got smaller, if anything, during past six months. Palpitations and shaking for six months. Perspiring for two months. Menses regular—scanty. No exophthalmos.

Fine tremor of hands. Coarse rhythmical tremor of feet, stopping for a time by voluntary effort and then beginning with renewed force; lateral as well as up and down movement. Sometimes general tremor of body; some of tongue.

Attitude and mask-like face resembling paralysis agitans. Reflexes normal.

(8) *Case of Encephalitis Lethargica.*

By Dr. BUZZARD.

B. D., aged 43.

Family history.—Father died of carcinoma of liver; brother has neuritis in his arms. Previous history: good, but patient "nervous."

September, 1920: Pain in both legs, which makes walking difficult, but soon improved.

November, 1920: Pain in right knee and weakness of right ankle; no numbness, but foot gets cold. No complaint of arms. Right foot "dropped" three weeks.

January 5, 1920.—Examination: Fibrillation of deltoids, pectorals, and triceps; more on left side. Weakness of sterno-mastoids and of above muscles. Slight weakness of extensors of wrists. Fibrillation of right quadriceps and wasting of all muscles of right leg: foot dropped. Some weakness of left leg; much weakness of right, especially of extension of foot and toes. No incoordination.

Sensation: Pain in right calf; deep pressure of muscles of legs painful; some loss of pin-pricks and heat and cold inner side right leg.

Reflexes: Arm and knee jerks increased, right ankle jerk absent, left present, no clonus. Abdominals brisk. Plantars: right flexor, left extensor.

(9) *A Case of Echinococcal Cyst of Left Parietal Region.*

By Mr. SARGENT.

M. E., aged 15.

April, 1920: Weakness of right hand, progressing to whole arm.

July, 1920: Peculiar feeling in right arm and face, starting in fingers and followed by vomiting.

September, 1920: Similar attack.

October, 1920: Right foot caught in walking.

December, 1920: Optic neuritis noted.

December 25, 1920: Pain in left frontal region; noises in ears; drowsy; vomiting; some defect of speech.

January 2: Slight hesitancy of micturition.

On admission into hospital, January 4, vision $\frac{5}{5}$, papillœdema, R. + 6D., L. + 5D. Right lower face weak, right hemiparesis arm and leg, slight loss of sense of position, localization of touch, and recognition of objects on right limbs. Pin, cotton wool, and vibration less distinct on right. Reflexes: deep, increased on right, abdominals less active on right; plantars, right extensor, left flexor.

January 14, operation: large cyst removed reaching surface just behind left fissure of Rolando, contained 60 c.c. of fluid, diameter about 6 cm. Pathologist's report: echinococcal cyst; fluid, albumin 0·015 per cent., sugar a trace; chlorides 0·57 per cent., sp. grav. 1·006.

After operation a few fits with twitching of right face, tasting movements of lips. Rapid general improvement. Subsidence of papillœdema.

(10) *Case of Spinal Tumour removed by Operation.*

By Mr. SARGENT.

A. W., aged 42.

June, 1918: Pain left side round iliac-crest.

June, 1920: Operation on left kidney, pain slightly improved, but soon recurred.

Three weeks later numbness of both legs, left foot dropped, pain reached left foot; soon right, also weak and painful. Slight hesitancy of micturition.

Examination: Spine not tender, but sitting up painful. Spasticity of both legs; some wasting; fibrillation of hamstrings and calf muscles. Spastic and unsteady gait, Reflexes: knee jerk very brisk: ankle jerk present. Plantars: double extensor. Abdominals absent below umbilicus. Sensation: pin-prick lost; L. 2 – S. 2 R.; D. 9 – S. 2 L.; sense of passive movement in toes lost. Cerebro-spinal fluid: yellowish fluid coagulating solid in tube; cells absent; albumin 3·70 per cent.; Wassermann reaction negative.

Operation, November 11, 1920: Tumour seen and felt through dura opposite cut arches of D. 11 and theca opened without wounding arachnoid; tumour sub-arachnoid, soft and encapsuled on posterior and left lateral aspect of cord; removed and theca sutured.

Pathologist's report: Myxomatous tumour with clumps of endothelial cells.

(11) *Case of Optic Atrophy from Neuritis and Gastric Attacks.*

By Dr. WILSON.

W. C., aged 56.

Family history: Father died of cancer.

Past history: Syphilis at 24.

1898: Sight impaired, treated at Moorfields in 1901 with mercury.

1902: Pain in stomach two hours after food, relieved by vomiting, improved since 1915 until recently. No unsteadiness of walking and no sphincter disturbance.

Present state: Visual acuity: right, $\frac{1}{60}$; left, $\frac{1}{60}$; on looking to right of fixation point, at that point vision almost nil.

Fields: Loss of upper half and general constriction, with central scotoma.

Fundi: Old atrophy. Argyll Robertson pupils. No ataxy. Deep pressure of calves very painful. Deep reflexes absent. X-ray suggests cicatrix from old ulcer or growth of stomach with delay in emptying of stomach. Wassermann reaction: negative in blood. Cerebrospinal fluid: no increase

of cells, globulin in cerebrospinal fluid. Blood count: red cells 3,700,000; hb. 70 per cent.; white cells, 10,500.

(12) *Case of Paralysis Agitans following Malaria.*

Shown by Dr. S. A. K. WILSON.

H. C., male, aged 35.

Malaria in England in 1918, blood tested at special malaria hospital (Connaught Hospital). A month later had "kicking" and twitching movements of left side, which improved after May, 1919.

In December, 1919, had weakness of left side and some slight stiffness of both hands. He began to stoop and to have difficulty in turning over in bed. By May, 1920, his right leg was also stiff. August, 1920: tremor, chiefly of right hand, slowness of speech and difficulty in opening mouth.

Examination: Slight weakness of left lower face. Spasm and rigidity of sterno-mastoids and trapezii. Tongue deviates to right. Mask-like face. Rigidity of trunk and proximal limb muscles. Pill-rolling tremor of hands. Paralysis agitans attitude and gait, with festination and retropulsion.

(13) *A Case of Epilepsy with Acromegaly and Unilateral Tremor.*

By Dr. J. TYLOR FOX.

W. G. O., born November 27, 1895. Nothing neuropathic recorded in the family. This patient's history and present condition may be classified under three headings; his epilepsy, his unilateral tremor, and his dyspituitarism.

Epilepsy.—First fit at fifteen months, second three or four years later; after that about every three months. Since admission to Lingfield Colony eight and a half years ago, he has had attacks of petit-mal every two or three months with epigastric aura, loss of consciousness without spasm lasting two or three seconds in all, also three major fits in the last two years.

Neurological signs.—Has had tremor of the left arm and leg "as long as he can remember." The tremor is continuous, apparently unaffected by voluntary movement, coarse, averaging five or six a second, and most marked in, if not confined to, the muscles about the large proximal joints, i.e., the shoulder, hip, knee and elbow. It ceases during sleep.

The left upper arm is $\frac{3}{4}$ inch less in circumference than the right. The forearms, thighs and calves appear to be equal on the two sides.

Reflexes, superficial and deep, give a normal response, though those on the left side are rather slower than on the right. Dynamometer readings 110 each side. No vasomotor differences between left and right side. No interference with sensation. No tremors of face, but coarse tremors of tongue. Optic discs, normal. Field of vision by rough perimetric tests, normal.

General signs.—Curvature of spine said to have been first noticed at fifteen months and attributed to rickets. Teething is also said to have started at fifteen months, walking and talking at eighteen months. Skeletal changes most

marked in skull and spinal column. Cranium tilted backwards on the facial portion of the skull. Superior and inferior maxillæ, large, especially inferior. Teeth pitted and grooved, and in upper jaw separated from each other. Nostrils, large and broad. Supra-orbital ridges, well developed. Eyelids, thick. Tongue, large. Well-marked kyphosis with scoliosis to the right in dorsal region. Lordosis in the lumbar region. The skiagram taken at the National Hospital, Queen Square, of the skull of this patient quite negative. The bones of the skull are normal in appearance, the sella turcica is of about normal size, its outlines can be well seen; the posterior clinoid processes are quite distinct, and the cavity does not encroach on any of the fossæ in front of it at all. As far as the skiagram goes, there is nothing abnormal to be made out. Right testis undescended and removed some years ago. Left testis very small. Thyroid not palpable. "Has never had a headache in his life." Temperature 97° to 98° F. Systolic blood-pressure, 110. Urine, daily quantity 49 oz., no sugar, no albumin. No diminished sugar tolerance. Patient is of fair intelligence, but his memory is poor.

(14) *Case of Torsion Dystonia.*

By Dr. BLANDY (for Dr. COLLIER).

H. R., aged 8, male.

History.—Six months after an attack of diphtheria (aged 5) involuntary tonico-clonic movements of an extensor type developed, with in addition perverse voluntary movements of the hands. The condition has shown marked remissions, but with each exacerbation has become more intense and more extensive.

First noticed in the right arm, with "loss of use of the hands," the movements gradually involved the neck, face, tongue, then left arm, trunk, and finally, in the severest stage, the legs.

February 1920, after being knocked down by a bicycle, there was a history suggestive of a tonic fit, followed by an exacerbation of extreme degree, associated with difficulty in eating, drinking and sleeping, and in starting the organic reflexes, owing to the excessive movements.

He could only sleep on his face, head extended, right arm tucked in behind his back.

Past history.—Nothing abnormal till aged 5, since when has stopped growing, and has had an intermittent morbilliform rash.

Family history.—Father has "bronchial asthma." Sister had chorea after rheumatic fever, maternal grandmother, mother, aunt, two uncles, had enlarged thyroids (the aunt died of Graves' disease).

Physical signs.—Small for age, intelligent; normal cranial nerves, sensation, reflexes. Wassermann reaction negative in serum and cerebrospinal fluid.

Motor system.—Hypertrophy of muscles most in spasm. Hypotonia during relaxation—no tremor nor ataxia, good power. Voluntary fine movements of hands awkward and slow. Hands in posture of flattened fists. Intermittent

clonico-tonic, tic-like, bizarre, involuntary movements, i.e., extension of head and shoulders with protrusion of tongue, &c. Extension of right arm with over-pronation of wrist, &c.

The child is at present date in a remission.

(16) *Cholesteatoma in Ponto-cerebellar Angle.*

Shown by Dr. J. G. GREENFIELD. (Dr. RUSSELL.)

Henry M., aged 27.

History.—Staggering gait, six months; left-sided deafness and headache—one month. Handwriting shaky, and difficulty in swallowing, a few weeks.

On examination: Papillœdema right and left. Coarse nystagmus to left, fine to right. Progressive nerve deafness on left side. All deep reflexes over-active. Double ankle clonus. Plantars upwards (Babinski) right and left.

Post-mortem examination three months after admission: Large cholesteatoma of left ponto-cerebellar angle, passing over the side of the pons under the basilar artery. Eighth nerve flattened, and internal auditory meatus enlarged and containing a prolongation of the tumour.

(17) *Colloid Ball-like Tumour in Third Ventricle, immediately over the Infundibulum.*

Shown by Dr. J. G. GREENFIELD. (Dr. RUSSELL.)

John G. C., aged 36.

History.—Headaches, worse when tired, sixteen months; diplopia, two weeks before admission. No vomiting.

On examination: slight papillœdema right and left; diplopia on looking to extreme left, from paresis of left external rectus. Left facial paresis.

Motor and sensory systems normal except for coarse tremor of outstretched hands.

Reflexes: all normal.

One week after admission he had several "fits" in which he rubbed the back of his head and was "queer" in his manner. He remembered nothing of them afterwards.

Post-mortem examination two weeks after admission: Ball-like encapsuled tumour in third ventricle causing hydrocephalus of both lateral ventricles. It is extremely tough after hardening in formalin, and feels like a solid rubber ball. Microscopically it is composed of colloid with a fibrous tissue capsule.

Notices of Recent Publications.

Recent Theories of Hearing.

The Analytical Mechanism of the Internal Ear. By Sir Thomas Wrightson, Bt., D.L. 254 pages + ix. London: Macmillan, 1918.

Under the above title Wrightson describes the mechanism of the sense of hearing, from the reception of the sound waves by the drum, to their arrival as auditory sensations at the brain.

The subject matter may be considered in the following separate sections;—

(1) The physical properties of sound.
(2) The ossicles and ear drum.
(3) The cochlea and its contents.
(4) The hypotheses of audition.
(5) Appendix by Keith on the anatomy of the ear.

Section 1.—The first part of this section, which is introductory in character, deals with the physical properties of sound, the formation of beats, summation and difference in tones and the like. The description is interesting, simply put, and does not call for special comment.

The ohmograph, of which a full description is given, does not appear to be novel except for a few mechanical details. The mathematical analysis of certain compounded sine curves which follows must now be considered in detail, since it forms the basis of the hypothesis of audition which Wrightson states later in his book. The process of analysis which Wrightson described may be briefly stated as follows: Three curves are prepared, the sine curve of a note A, the sine curve of another note C, and the curve obtained by summing both of them by the ohmograph. A pair of callipers is now taken, set to the wave length of the note A, and then tried on each crest, trough and crossing point of the compound curve in turn; if the two feet of the callipers fit any of the distances on the compound curve, such distances are marked A1 and A1. The next pair of points found to correspond to the distance between the feet of the callipers is marked A2 and A2 and this process is repeated until one complete cycle has been examined. In one case three such distances were found, and in another four. If now the whole process be repeated with the wave length of the note C, then again a number of distances between crests, troughs and crossing points in the compound curve are found, which correspond with the wave length of the note C. In one case five such crossing points were found and in another three. If finally the above process be repeated with a curve compounded of three or four different wave length sine curves,

then as before a number of distances are found on the compound curve, which are equal to those on each of the constituent curves.

Wrightson now goes on to show that if the wave length of the summation and difference tones be taken, then as before distances equal to these are found on the compounded curve. Wrightson draws the conclusion, therefore, that when two or more musical notes are sounded together, the compound curve contains a number of "impulse points," the time intervals between certain of which represent those occurring in both the constituent musical notes and also in their summation and different tones. Wrightson points out later that it is the recognition of these time intervals by the brain which enables us to recognize these different constituent tones.

Wrightson now goes on to state (p. 30) that no mathematical exactness is postulated between the time periods in the compound sound waves and those of its constituents, yet they may be sufficiently accurate for the theory, "as the ear, in judging of musical intervals, does not depend upon absolute accuracy of vibration ratio, a fact well known to practical musicians." This want of exactness is readily detected in the diagrams which Wrightson gives; thus, taking the first (fig. 3) as an example, it will be found that the distance A_2-A_2 is smaller than the wave length of A, while A_3-A_3 is larger.

Boring and Titchener[1] have pointed out that taking the above analysis at its face value the following criticisms can be advanced against it. (*a*) The summation and difference tones are both an octave too low. (*b*) Besides the pure tone the ear should also hear according to Wrightson the octave below it; this the ear certainly does not do. (*c*) It should also hear the octave above it, and this again is not the case. The reader will conclude from this that even if the fact is ignored that the mathematical analysis is not exact, it still further prejudices itself by leading to false deductions. The proof must now be given that it is the analysis itself which is at fault; supposing that the compound curve in fig. 3 be again turned to and that the callipers be set not to the wave length of either A or C, but to a purely arbitrary distance somewhere between these two values. Then if this distance be laid off as described above, it will probably be found that it is several times repeated in one cycle. I performed the process for twenty arbitrary distances and found each of these distances represented some three or four times in the compounded curve. This at first sight astonishing result was partly explained by finding that the total number of distances in one cycle varying between 1·5 mm. and 44 mm. in length was approximately 400. Assuming that all distances are equally represented, each distance would vary from the next by approximately 0·1 mm. If we assume an inaccuracy of setting of say 0·1 mm. then any arbitrary distance between 1·5 mm. and 44 mm. will be represented on the average three times (one exact fit, one 0·1 mm. too large, and 0·1 mm. to small).

We thus see the explanation of Boring and Titchener's remarks. "It is strange" they say, "that the theory, if we take the diagrams at their face

[1] *American Journal of Psychology*, April, 1920, vol. xxxi, pp. 101-113.

value, should thus insist on proving itself. Moreover, there is something disconcerting about a theory that will come right on any assumption." This mathematical foundation that Wrightson had laid for his theory therefore fails since the brain cannot be expected to pick out certain time intervals, and construe them as musical notes when apparently all time intervals between certain limits are equally represented,

Section 2 contains a well illustrated description of the ossicles, their suspensions and musculature and the way in which they function. Wrightson now attempts to calculate on "static" principles the total increase of effective pressure due to the lengths of the ossicular levers and the areas of the drum and the piston of the stapes. It is clear to the reader, however, that the calculation should be a "dynamic" one, since the levers when functioning are in rapid motion. For not only must work be performed in setting and keeping the ossicles in vibration, but also in overcoming friction at the joints between them, and the damping action of the air surrounding them.

Section 3 contains a description of the cochlea and of the organ of Corti. The anatomy and histology of the parts concerned is well described, and easily understood. When, however, Wrightson commences to describe the calibration of the cochlea the argument becomes very difficult to follow; for the ideas are confused (power, work, force, &c., being used synonymously as Boring and Titchener have pointed out). Further, Wrightson's final conclusions appear to be in actual error, for he claims that whereas when air is transmitting sound waves the air particles come to rest twice during each wave (at each crest and at each trough), the fluid of the cochlea, on the other hand, comes to rest four times, not only at each crest and each trough, but also each time the air particles pass through the mid position of their movement, i.e., each time the sine wave, representing the motion, crosses the axis. This conclusion, as Boring and Titchener point out, is at variance with the third law of motion, that action and reaction are equal and opposite. In view of this error of Wrightson, the remarks of Keith in reference to this matter become almost pathetic: "I had," Keith writes, "foolishly regarded the crests and troughs of sound tracings . . . as the points at which stimuli were produced, for at those points the ciliary movements appeared to be reversed. But I could not see how stimuli could be produced at the crossing points, until it was made clear to me by Sir Thomas Wrightson that the crossing points in the tracing of a sound wave are points at which there are equally great changes in pressure and movement." Yet Keith's original view was the correct one, and the view that Wrightson substituted was the incorrect.

Section 4 contains Wrightson's hypothesis of audition, which may be stated as two separate propositions as follows:—

(*a*) That when sound waves enter the ear, the whole of the basilar membrane is set into motion, and the different parts of the membrane move synchronously, rising and falling together by the same amount (p. 104). These movements of the basilar membrane cause to-and-fro movements of the hair-cells, relative to the membrane tectoria above them, and these at each change in direction of the movement cause impulses to pass up the auditory nerve.

(*b*) That the impulses passing up the auditory nerve occur four times during each complete air vibration, i.e., at each crest, trough and crossing point; and further that the time intervals between these impulse points are in certain cases approximately the same as those which occur in the individual tones which together are causing the air to vibrate. It is the recognition by the brain of these time intervals which causes the appreciation of the individual tones in a musical chord, the appreciation of overtones, harmonies and summation and different tones being similarly recognized.

About this second half of his theory (*b*) it may be said that it is this part which individualizes Wrightson hypothesis from other displacement hypotheses which have preceded it, e.g., from Rutherford's telephone hypothesis; but it is also this part which must be abandoned, since, as I have shown above, its supposed basis does not actually exist. The remaining features of the hypotheses are so like those of Rutherford's that the two must stand or fall together. Both hypotheses hold that the impulses travelling up the auditory nerve correspond in a definite manner in their time relationships and intensities to the air waves which set them up; both believe that the basilar membrane lacks any power of analysis; both relegate the ultimate analysis to the brain. The criticisms advanced against Rutherford's theory in the past therefore apply with equal force to Wrightson's, namely, that the initiation of nervous impulses at a rate which may reach 40,000 per second and their conduction up the whole length of the auditory nerve and through two different sets of synapses, and their final enumeration by the brain, are all processes which tax the credulity of the physiologist to the utmost. The classic experiments by which Rutherford sought to give his hypothesis something more than an imaginary basis are too well known to require description here; the grafting on to that basis of an additional hypothesis, as Wrightson has done, without at the same time any experimental extension, would seem to be a mistake.

Section 5 consists of Keith's appendix on the histology and morpho-anatomy of the organ of hearing, and comprises nearly half the book. For this essay I have nothing but admiration; it is absorbingly interesting, well written and full of that subtle knowledge that we should expect from the pen of a great anatomist. Whereas Wrightson disappointingly fails us at just those points where we should have expected an engineer to have made the surest guide, Keith serves us admirably not only when dealing with his own subject, but also when he digresses to physics and mathematics. If one has any single feeling which one would wish was not there, it is the regret that Keith had heard of Wrightson's hypotheses. Excellent as Keith's essay is, might it not have been better still with possibly not only Wrightson's hypothesis but also all others left out, a simple description of the parts of the inner ear without any idea in the background of hypotheses? Having considered above the different sections in turn, it remains to appraise the book as a whole. It is a book which in my opinion all interested in hearing should read; it contains one of the best descriptions of the comparative anatomy of the cochlea that I know, it contains many facts about the organ of Corti

which are not to be found elsewhere. We have Keith personally to thank for many of them. To Wrightson I would wish to say that although it has been my lot to criticize his hypothesis, yet I would like to express my admiration for the facts that he has brought to light about the organ of hearing and the clever way in which he has described them in his book.

H. HARTRIDGE.

The Psychology of Sound. By HENRY J. WATT, M.A., Ph.D., D.Phil., Lecturer on Psychology in the University of Glasgow. Pp. vii + 241. Cambridge: University Press, 1917.

This book is not very well named. It would be better described as: A theory about the nature of psychology illustrated by speculation concerning the character of auditory experience. Watt's first conviction is that psychology must occupy a position wholly independent of physics or physiology. His second is that all forms whatever of sensory experience must conform to a single scheme, and must illustrate the same structural principles. His third is that occupying an exceedingly important position among the attributes of sensory experience are what he calls "order," and something else of the nature of what other writers have called "extensity." It is on these assumptions, which are perfectly general, and are obtained by theorizing, rather than by experiment, that his account of audition is based. So, in the last chapter, we find him saying with the greatest honesty: "The theory expounded in this book has been devised by the writer not in the interest of a study of hearing as such, but for the sake of the more inclusive study of the whole range of sensory experience. This work on hearing is thus only an episode in a larger effort. It is my firm belief that all our sensory experiences can be completely accounted for in general systematic terms without our having recourse to the discoveries of physics or physiology at all." And what is written in words here is written in the spirit throughout the whole book.

An adequate review of Watt's volume ought therefore to begin by a thorough examination of his underlying assumptions. This cannot here be attempted, but one thing must be said. Whether in this realm or in any other, Watt cannot expect to gain many adherents to his views unless he will express his contentions in language more clear to the ordinary understanding.

Take the conception of "order" as an attribute of sensation, and thus of sound. It is introduced at the beginning of the book (p. 8). It occurs frequently all the way through to the very end (p. 215). Together with that of "volume," which is the name for the "extensive implication" of sounds, it bears all the burden of the theories put forward concerning tone, noise, fusion, consonance, the localization present in binaural listening, and many other matters. Yet, in spite of this, it remains throughout an elusive conception. The "ordinal differences of the elements of hearing," for example, "are the basis of the binaural localization of hearing" (p. 177), and "there is no

primary difference either physiologically or psychically between the binaural basis of localization and the uniaural basis of pitch" (p. 192). But pitch, which is thus a "kind of order," certainly is not to be supposed to be spatial (see pp. 28-29, 213, &c.), whereas localization, which is also "a kind of order," as certainly is. "Spatiality" is said to have "emerged" from "something in the single ear," this "something" being the ordinal, non-spatial attribute of the sound sensation. How it "emerged" is not clear, although the process is described as one of integration "with those ordinal differences of other senses that are dependent upon the physical positions of their stimuli."

In any case what "emerges" from the argument would appear to be that there are at least two kinds of order, one spatial and one non-spatial. Perhaps there may be any number of other kinds also. Perhaps, in point of fact, "order" as an attribute of all sensory experience simply means that within any such experience differences of some sort occur, and may form a basis upon which all kinds of subsequent discrimination proceed. Thus on p. 213 "order" is definitely spoken of as a "purely psychical arrangement." It would appear that material of *any* kind whatever might be so arranged. Positions, or moments, or qualities might all be "ordinal"; but it could not in the least be inferred from this that they were alike in any other respect than that they were an "arrangement." If this were literally all that was meant, although both pitch and localization could be spoken of as a "kind of order," it would by no means follow that the second "emerges" from the first. Nothing would follow, in fact, except that they would both be a kind of arrangement. This appears clearly to be inadequate. And it helps to show that in so far as the concept of "order" is to be of value in the solution of any particular problem ordinal differences must be treated as having a specific nature.

It is difficult to be certain about any of Watt's meanings. But it seems highly probable that "order," as belonging to sound, really signifies a good deal more than a mere arrangement. As it is actually made use of, the conception appears to be derived from a belief that tones are "volumic masses." That is, they are spread-out arrangements in which the various "atoms" have relative positions, and in which the "central atom predominates, thus giving the whole a pitch" (p. 206). Tone is a balanced mass of such atoms, noise an unbalanced mass. All problems of fusion are simply a question of the coincidence of volumes, and their resultant balance; while interval is mainly a question of the proportion between different parts of a "volumic outline or stress." Add "motion" to order and volume, and to Watt the structure of melody becomes clear. Study of the formation of scales brings us back to the same attributes combined and modified in various ways. As others have remarked, Watt's whole theory rests on his conception of volume, and this conception is wholly speculative, having no empirical basis that can be discovered. Supposing now that it is right to treat order as involved somehow in volume, then, as the latter has avowedly extensive implications, it seems likely that, however much Watt may deny the fact, in some way or other a spatial character does lurk in order as the latter is here

attributed to sound. This would help to explain why Watt is certain that pitch differences are not qualitative, and might help to render the treatment of localization a little more intelligible. But whether in truth such a spatial character does belong to auditory sensations would still be a matter for dispute.

There is in the book a long chapter on "Physiological Theories of Hearing," in which summaries of all the leading theories are presented. These are of very considerable interest, though they are not always sufficient. Watt himself rejects the resonance hypothesis, as would be expected. He supposes that the basilar membrane is exceedingly elastic, and that to hear a sound means that an actual wave form is set moving along the membrane. A "physical tone produces a wave of depression of the basilar membrane beginning from the basis and extending along in proportion to its pitch, with a point of maximal depression in the centre round which relative intensities are arranged symmetrically and decreasingly." There is no evidence that this actually does happen. But that does not matter. For, experience being what it is—or what Watt alleges it to be—the assumption of the supposed physiological mechanism is perfectly consistent. If any investigation should be able to show that the structures involved could not react in the way proposed, then the investigation must be wrong. This is where the "independence" of psychology comes in.

The book shows upon every page evidence of the most careful research. But it is research by reading and thinking, not by experiment. Now every experimentalist in psychology knows that much of what chiefly convinces him is hard to put down on paper. And any man who relies mainly upon a critical reading of experimental work may fail to be convinced because some only of the data are set before him. In any specific psychological problem it is just as important for theorists to conduct experiments as it is for experimentalists to construct theories. In spite of his enthusiasm for his task, of the thorough way in which he has covered the field, and of the air of complete certainty with which he publishes his most interesting views, Watt must be said to have written a somewhat one-sided treatise. It is, in fact, philosophical rather than psychological. Perhaps it is consistent with all the facts. But a theory which no facts can confute may be of as little worth as one with which no fact can be made to agree.

F. C. BARTLETT.

The Form and Functions of the Central Nervous System. An Introduction to the Study of Nervous Diseases. By FREDERICK TILNEY, M.D., Ph.D., Professor of Neurology, Columbia University, and HENRY ALSOP RILEY, A.M., M.D., Associate in Neurology, Columbia University. Pp. 1,020, with Glossary and Index. New York: Paul B. Hoeber, 1921.

Within recent years no branch of medical science has made more rapid progress than neurology. The work of investigators on the normal and

abnormal histology and physiology of the nervous system in men and animals has led to the accumulation of a vast amount of knowledge, which is scattered widely throughout the literature of different countries and is not readily accessible. There is therefore increasing need for the co-relation and presentation in book form of this wealth of data and of the main speculations which have arisen from them upon the structure and functions of the nervous system. It is this task that the authors have attempted; but unfortunately the result of their labours has not been followed by the success that might have been hoped for.

They say in the preface that their book is intended for the use of students, as an introduction to the study of nervous diseases; but, as it contains upwards of a thousand pages of text, it is obviously much too large for this purpose. What is required by the student in his medical course is a clear and concise account of nervous structure and function presented in a form in which it can readily be applied to the study of disorders of the nervous system. It should not be encumbered with debatable points of detail or an ambiguous and misleading terminology. As far as possible it should give the facts and show how they can be united by accepted general principles.

If, on the other hand, the book is to be regarded as a comprehensive treatise for reference, as its size might indicate, it falls short in many ways. For example the bibliography is never referred to in the text, a want which is all the more felt as the authors tend to dogmatize on controversial questions. The perpetual insistence on syndromes is trying to the reader, who is anxious to discover the significance of some sign or symptom. For instance "the Babinski reflex" is repeatedly mentioned, but there is no coherent or systematic account of current views as to its nature and origin. The term "sensory" is employed to designate both sensory and non-sensory afferent fibres and impulses, a usage which inevitably gives rise to considerable confusion. The information given is extensive, but is insufficient on several important points both of structure and function. Thus, the fasciculus retroflexus of Meynert is scarcely mentioned and vision and speech are inadequately dealt with. To illustrate the disorders of function, which result from lesions of different parts of the nervous system, the authors have drawn freely from their clinical records; but the value of these examples is often diminished by the incompleteness of the observations and, occasionally, by the diffuseness of the morbid changes.

The meaning is not infrequently obscured by the use of barbarous terms such as "arthresthesia," "piezesthesia," &c., which are unnecessary and have not received general acceptance. The ear of the reader is jarred by "juxtagriseal," "behavioural" and "tractability" and the proofs have been carelessly read. "Deiters" and "Gowers" are correctly spelt in the index, but are habitually wrong in the text; the name of the French neurologist becomes "Déjérine" in the index, "Djerine" on three of the plates, but is rightly printed in the references.

Of the 591 figures, containing 763 illustrations, many are good, but others,

especially the reproductions of photographs of sections of the nervous system, could be considerably improved.

We have been somewhat lavish in criticism because we believe that an authoritative and comprehensive presentation of the available knowledge on the applied and comparative anatomy and physiology of the central nervous system is much needed. This book will probably pass into a second edition, and if so there is no reason why it should not be greatly improved to meet the want.

Manic-depressive Insanity and Paranoia. By Professor EMIL KRAEPELIN, of Munich, translated by R. MARY BARCLAY, M.A., M.B., from the eighth German edition of the "Text-book of Psychiatry." Vols. III and IV. Edited by GEORGE M. ROBERTSON, M.D., F.R.C.S.Edin., Professor of Psychiatry in the University of Edinburgh and Physician to the Royal Asylum, Morningside. Pp. 280. Edinburgh: Livingstone, 1921.

To Professor Kraepelin is due the credit of recognizing manic-depressive insanity as a single morbid group characterized by profound alterations in emotional tone, but unaccompanied by progressive mental deterioration. The phases of exaltation or depression may be of very different intensity; thus a patient, who suffers from severe recurrent attacks of melancholia, may show symptoms of hypomania so slight that they are considered as the natural rebound of convalescence. Conversely, a man who becomes excited and troublesome, and on recovery passes into a quiet unhappy state, may appear to be brooding over the consequences of his outburst of folly. But the most difficult cases from the point of view of the neurologist are those usually called "recurrent neurasthenia"; each attack is attributed to some worry or physical strain, and the essential nature of the condition and its close relation to manic-depressive states is not recognized. Careful inquiries will, however, elicit the suddenness of the onset and its want of direct dependence on emotional shock. This morbid condition persists for many months or even years and then passes away as suddenly and apparently as causelessly as it came.

We therefore welcome this translation of the chapters in Professor Kraepelin's book dealing with manic-depressive states in all their forms, straightforward circular insanity, recurrent excitement or depression, repeated attacks of "neurasthenia," or mixed psychical conditions in which all these forms coexist to a greater or less degree. No cases are described, but each chapter is illustrated by a multitude of instances, which tend somewhat to obscure the general clinical picture. Every aspect of this protean malady is considered systematically: but at the end Professor Kraepelin confesses that he is completely ignorant of its true nature and cause.

The last two hundred pages are devoted to an account of Paranoia, in the narrower meaning of the term, as a primary form of insanity, which develops insidiously and leads to no permanent psychical weakness. Here, in these

chapters more clearly than elsewhere, we see the difficulties produced by a classification built up solely on overt symptoms and deviations of conduct.

The translation is accurate but clumsy: the book is full of German phraseology and it is a pity that some of the legends on the figures have been left in the language of the original. English students who require a translation of so well-known a work may be puzzled by such explanations as "Gesunde richtig, Gesunde falsh," "Zornige Manie," "Depression mit Reizbarkeit," and the numerous long German citations.

Anxiety Hysteria. Modern Views on some Neuroses. By C. H. L. RIXON, M.D., M.R.C.S., and D. MATTHEW, M.C., M.B., Ch.B. Pp. 124. London: H. K. Lewis, 1920.

The authors have attempted to express in simple language their views upon the psychopathology, symptomatology, diagnosis and treatment of the more common forms of the psychoneuroses of war. They address especially the general practitioner who, as a medical referee or a member of a medical board under the Ministry of Pensions, has frequently to deal with such patients; and as a practical guide to investigation and to rational methods of treatment on broad lines it will serve a useful purpose.

A chapter is devoted to psychopathology and some of the mental processes concerned in the production of symptoms are explained. Diagrams are freely used and will perhaps be found helpful, although they are apt to lead to somewhat stereotyped notions of the processes they are intended to elucidate. The views expressed agree with those of most observers, that the conflicting forces at the base of war neuroses are those connected with the instinct for self-preservation and with the sentiments linked round the ego-ideal. The terminology employed is as a rule clearly defined but it would have been advantageous to have used separate terms for voluntary and involuntary forgetting.

Psychology and Psychotherapy. By WILLIAM BROWN, M.D., D.Sc. Pp. 196. London: Edward Arnold, 1921.

This little book deals in an easy and popular manner with the various aspects of modern psychopathology. The introductory chapters are devoted to dissociation and to the theories evolved to explain hysteria and the functional neuroses. The second part contains a clear statement of the teaching of Freud, with a short description of psycho-analysis and word-association. This is followed by a chapter on psychotherapy and a section on the lessons of war. The style is clear and simple and the book can be recommended to those who are anxious to know something of the diverse views now current on the processes responsible for the psycho-neuroses.

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EDITOR.